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ACETYLCHOLINESTERASE ACTIVITY IN THE COCHLEA.*

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Since the discovery of "der Vagusstoff" by Loewi[1] and the identification of this material as acetylcholine by Dale[2], problems in acetylcholine distribution and function have been investigated intensely by physiological and biochemical means. Through recent refined histochemical techniques designed to show discrete localization of acetylcholinesterase, knowledge of the distribution of this enzyme has been greatly advanced. The number of sites in the nervous system known to contain acetylcholinesterase has been increased almost to the point where this enzyme might be considered to be universally distributed in the nervous system, as Nachmansohn[3] contends; yet it is clear that the quantities of acetylcholinesterase differ widely in different neural structures. Acetylcholinesterase was thought to be absent from sensory or afferent neurones[4] until recently when Koelle[5] demonstrated that low concentrations of the enzyme do exist at least in several sensory ganglia and nerves.

It is held that the presence of acetylcholinesterase in the nervous system is strongly presumptive evidence that the

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cholinergic mechanism operates where the enzyme is found. Acetylcholinesterase concentrations in any given region of the nervous system have, in general, been found to parallel concentrations of acetylcholine[6,7] and cholineacetylase[8], indicating that these three substances occur together. The part played by acetylcholine in the transmission of nerve impulses along axons has not yet been completely elucidated. It is contended by some that acetylcholine is essential for the propagation of nerve impulses, while others hold that the acetylcholine acts only at certain cholinergic nerve endings.[9]

The purpose of the present experiment has been to determine whether or not a cholinergic mechanism is present in the cochlea. If acetylcholinesterase were found in the cochlea, it was hoped that the substance might be localized to particular sites, and that from these data, inferences might be drawn about cochlear function.

Sites of cholinesterase activity were looked for by using the method of Koelle[10,11,12,13]. This technique, in brief, consisted of incubating tissues in a solution of acetylthiocholine* in the presence of cupric ion. At points of acetylcholinesterase activity, the substrate was hydrolyzed, and the thiocholine liberated formed a precipitate of copper thiocholine. This substance was later converted to copper sulphide when the specimen was treated with ammonium sulphide; thus, copper sulphide, a rather stable and visible precipitate, was seen where acetylocholinesterase had been acting. While the choice of acetylthiocholine for the substrate biased strongly the activity of the cholinesterases in favor of acetylcholinesterase, it was considered wise to verify the type of cholinesterase found by treating some samples with diisopropyl fluorophosphate (D.F.P.)† so that non-specific (pseudo or butyl) cholinesterase would be inactivated; also, for further proof samples were incubated in a solution of butylcholine, some with and some without, D.F.P. treatment.

METHOD.

The experiments were performed on eleven cats and two guinea pigs. The animals were anesthetized by intraperitoneal injection of sodium pentabarbital (Nembuta then decapitated, and the cochleae were removed as possible. The calvarium was opened with a ro brain extricated, and the petrous bones we m large bone-cutting forceps. The cochleae were fr surrounding bone with fine bone-cutting forceps About one half of the cochlear wall was removed by fracturing the outer cochlear wall with fine bone-cutting forceps, so that the modiolus and cochlear coils were exposed to view. The scala media was opened throughout a large part of the exposed region, because the spiral ligament separated from its attachment to the basilar membrane and thus was removed with the outer bony wall. Frozen skeletal muscle was sectioned at a thickness of 15 micra and treated with the cochleae. A cochlea and muscle section were treated to demonstrate acetylcholinesterase activity, while another such pair of tissues was treated identically, except that it was not exposed to the substrate. In some experiments such pairs were also incubated in butylthiocholine with and without previous D.F.P. treatment.

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* Acetylthiocholine was supplied through the courtesy of LaWall & Harrisson Co., 1921 Walnut Street, Philadelphia, Penn.

† D.F.P. was supplied through the courtesy of Dr. Wm. H. Summerson of the Army Chemical Center, Edgewood, Md.

Pieces of fractured cochleae were first placed in a storage solution consisting of 9.0 ml. of 40 per cent sodium sulfate and 6.0 ml. water, kept at a temperature of 33°-35° C. As soon as possible the tissues were transferred to Coplin jars, which contained the incubating solutions in which the tissues were kept for 45 minutes at 38° C.

The acetylcholinesterase incubating solution consisted of 9.0 ml 40 per cent sodium sulfate, 0.6 ml. copper glycine, 1.5 ml. sodium acid maleate, 0.6 ml. magnesium chloride, and 2.1 ml. water. To this solution, prepared 15 minutes before the time of use, was added 1.2 ml. of acetylthiocholine solution. This was prepared by placing 23 mg. of acetylthiocholine iodide in a centrifuge tube with 1.2 ml. water and 0.4 ml. of 0.1 M copper sulfate. After centrifuging the copper iodide down, the supernatant fluid was decanted into the incubating solution, which was then filtered just prior to use; also, before the tissues were transferred to the incubating solution a trace of copper thiocholine was added.

Control tissues were placed in a solution containing 6.0 ml.

of 40 per cent sodium sulfate, 0.4 ml. copper glycine, 1.0 ml. sodium acid maleate, .04 ml. magnesium chloride, 1.4 ml. water and a trace of copper thiocholine.

At the end of the incubating period, all tissue samples were placed in 10.0 ml. 40 per cent sodium sulfate and 10 ml. water with a trace of copper thiocholine for five minutes. They were then transferred to 5 ml. 40 per cent sodium sulfate and 16 ml. water with copper thiocholine for one minute. They were next passed through water containing a trace of copper thiocholine for one minute. After this, the tissues were developed in a solution of 1.0 ml. saturated ammonium sulfide solution diluted with 30 ml. water which had been saturated with copper sulfide. The specimens were then rinsed in water for one second and were placed in 10 per cent formalin saturated with copper sulfide. The tissues which were treated to exclude non-specific cholinesterase were first of all placed in 1.5 ml. of one millionth mole D.F.P. with 4.5 ml. water and 9.0 ml. 40 per cent sodium sulfate for 30 minutes at 35° C. Frozen sections of striated muscle, 10-15 microns thick, were placed on slides, allowed to dry one minute, and were then run through the same process.

Those specimens which were treated with the substrate butylthiocholine were divided into two groups as to whether or not treated with D.F.P. Those that were not treated with D.F.P., and thence were run to show non-specific cholinesterase, were first placed in a storage solution composed of 10.5 ml. 40 per cent sodium sulfate and 4.5 ml. water. The incubation solution for these tissues was composed of .6 ml. copper glycine, 1.5 ml. sodium acid maleate, .6 ml. magnesium chloride, 10.5 ml. sodium sulfate, .6 ml. water, 1.2 ml. butylthiocholine, and a trace of copper thiocholine. D.F.P. treated specimens used as controls were placed in an incubation solution containing .4 ml. copper glycine, 1.0 ml. sodium acid maleate, 6.4 ml. magnesium chloride, 6.0 ml. sodium sulfate, 1.4 ml. water, .8 ml. butylthiocholine and a trace of copper thiocholine. The butylthiocholine was prepared by placing 43 mgms. butylthiocholine iodide, 1.8 ml. water, and .6 ml. of .1 molar copper sulfate in a centrifuge tube, the filtrate being saved and used.

Reagents were composed as follows:

Copper glycine: 3.75 gm. glycine, 2.50 gm. $CuSO_4 \cdot 5H_2O$, q.s. 100 ml.

Maleate Solution: 9.60 gm. NaH maleate, 52.2 ml. *N* NaOH. q.s. 100 ml.

Sodium Sulfate: 40 per cent weight per volume Na_2SO_4 adjusted to pH 6.00 at 38° C.

Magnesium Chloride: 9.52 gm. $MgCl_2$, q.s. 100 ml.

Copper thiocholine was obtained by filtering the incubation solution after use and storing for two to four days. The precipitate was collected by filtration.

After fixation, the cochleae were placed in a shallow bath of Ringer's solution containing copper sulfide. The tectorial membrane was removed under the dissecting microscope, and pieces of the organ of Corti and osseous spiral lamina were teased from the cochlea and mounted on slides in Apathy's gum arabic. Some cochleae were counter-stained, embedded in paraffin and sectioned; others were gold-toned, decalcified, embedded in celloidin and sectioned.

RESULTS.

All acetylthiocholine treated cochleae contained a dark stripe lying in the region of the organ of Corti, which was seen clearly with the naked eye (see Fig. 1). In no experiment did the controls have this precipitate on gross and microscopic examination. When examined under the dissecting microscope it was seen that the dark stripe faded away as the cochlear duct entered the unopened regions of the cochlea. Under high magnification the stripe in most cochleae was resolved into two parallel bands, the inner band being much more intense than the outer band (see Fig. 2). The precipitate was seen in all turns of these cochleae, but no attempt was made to study the relative density of the precipitate in the several turns.

While variable accessibility of chemical solutions to different cochlear regions would seem to be important in evaluating the relative densities of precipitate in the different cochlear turns, no such factor should play a role in the comparison of precipitate density of inner and outer bands, particularly

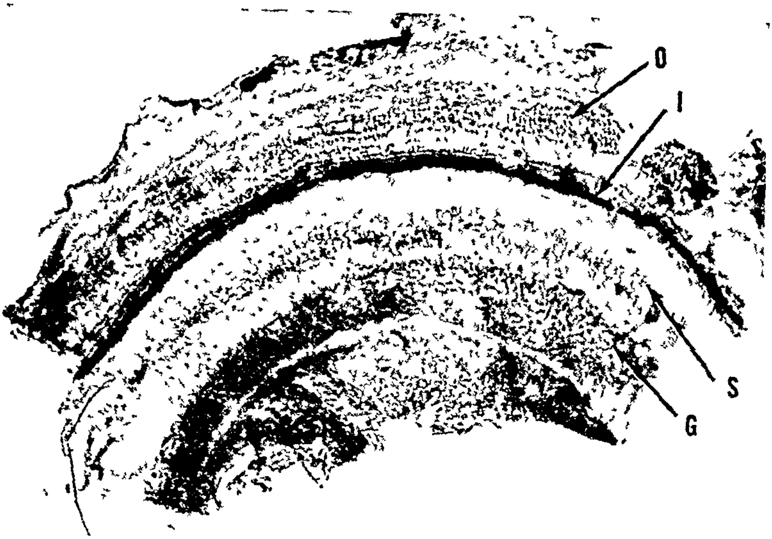

Fig 3

screte boundaries, was so broad and dense that adjacent
tological detail was largely obscured. The outer band con-
sted of small patches of precipitate arranged in rows. These
atches were ovate to cupulate in form, measured 2-5 micra
diameter, and lay in close relationship to the rows of outer

hair cells The precipitate was limited to areas consistent in position and size with the chalices of the hair cells A dense amorphous mass was seen lying in the region between the habenula perforata and the inner hair cells, extending beyond the confines of the hair cell chalices or nerve end-

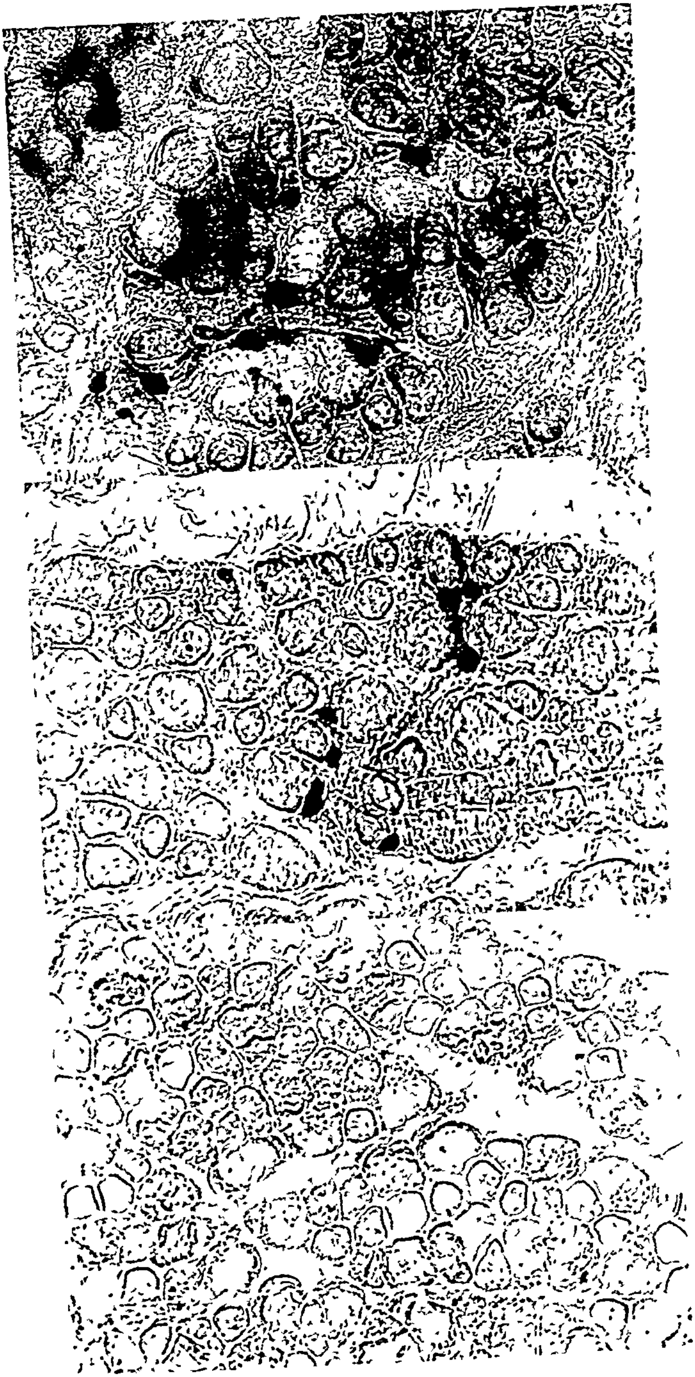

Fig 5

ings. In some areas fine lines of precipitate appeared to outline individual nerve fibers (see Fig. 4). These outlined fibres were most numerous near the habenula perforata, becoming sparser near the spiral ganglion. Since deeper parts of the specimen, such as the spiral ganglion, were not exposed adequately to the chemical solutions, the experiment does not provide information about these structures.

The muscle sections provided a check on each experiment to prove that the proper technique had been followed. In the muscle treated with acetylthiocholine the motor-end plates contained a dense precipitate, whereas in the muscle not treated with the substrate, the motor-end plates contained no precipitate (see Fig. 5). The density of precipitate bands in the organ of Corti and of the motor-end plates was not altered by incubation in D.F.P.; however, surrounding tissues were paler than non-D.F.P. treated specimens. This suggests that the slightly brownish discoloration of the specimens not treated with D.F.P. was due to the action of non-specific cholinesterase and thioesterases.

DISCUSSION.

The heavy brown amorphous precipitate found in the organ of Corti was believed to be copper sulfide found at sites where acetylcholinesterase had been hydrolyzed by acetylthiocholine. That these deposits were the results of acetylcholinesterase activity rather than from activity of non-specific cholinesterases or thioesterases, was borne out by the results of treatment of control specimens with butylthiocholine and D.F.P. Occasionally needle shaped crystals were seen in locations of high acetylcholinesterase activity. These were thought to be copper thiocholine[14] which had escaped complete conversion to copper sulfide, indicating that in these regions acetylthiocholine had been hydrolyzed by acetylcholinesterase. It was concluded that acetylcholinesterase was present in the organ of Corti intimately associated with the hair cells and their nerve endings.

According to the findings presented here, acetylcholinesterase found in the organ of Corti may be related to the hair cells, endings of the afferent neurones, or endings of the efferent fibres from Rasmussen's bundle. Derbyshire and

Davis,[15] in 1935, considered the possibility of a chemical mediator in the conversion of sound energy to acoustic nerve action potentials. They calculated that the latency of 0.5 to 0.7 msec. between the microphonic cochlear response and onset of the spike of the action potential was of an order comparable with the rates of release, action and destruction of acetylcholine.

Also neural summation for high frequencies might be explained by a chemical transmission hypothesis. For frequencies of 2000 cps and lower each volley of nerve impulses, as revealed by the action potential, can be clearly associated with one sound wave or another, but when the frequency is higher, *e.g.* 8000 cps, the nerve responds to a tone pip as a whole instead of to individual waves[16]; thus, the stimulating effect seems to be integrated by some process, termed "summation." in which the nerve responds as a whole instead of to the individual waves. The explanation might be that a chemical or enzyme reaction occurring at the sensory-neural junction is so rapid that for low frequencies the chemicals are deposited and destroyed before the onset of the next stimulating wave. The decay time is too slow, however, for it to be completely destroyed in the time interval between waves of a high frequency pip, so that a build-up would occur until adequate to trigger the action potential.

Gisselsson[17] recently made a searching review of the chemical mediator theory and confirmed the finding of Martini[18,19] that there were cholinesterases in these fluids. He obtained cat perilymph and endolymph by pipetting the fluids out through punctured round windows and basilar membranes. He then incubated measured volumes of the fluids with acetylcholine of known concentration, and compared contractions of leech dorsal muscle and frog rectus muscle bathed in the solutions with contractions of the indicator muscle in acetylcholine of known concentration. By this procedure values for cholinesterase activity were obtained.

Although acetylcholinesterase has been found in sensory neurones by Koelle[5], the concentrations were evidently quite low. The presence of such a conspicuous amount of the enzyme in the organ of Corti suggests that it may be related to the efferent nerve endings rather than to the hair cells of acoustic nerve endings. The efferent fibers were first described by Rasmussen[20,21] and called by him the "olivo-cochlear tract. The cells of origin of this tract lie medial to the superior olivary nucleus and dorsal to the nucleus of the trapezoid body in the medulla. The tract decussates hard by the colliculi of the facial nerve and passes into the inferior division of the vestibular nerve and then by three fascicles in a spiral course up the cochlea. The termination points of these fibers have not been discovered, although experiments of Portmann[22] and of Schuknecht[23] suggest that they end upon the inner hair cells. At any rate, it is quite certain that these fibers do not end upon blood vessels, or structures apart from the organ of Corti.

Experiments by Galambos[24] where amplitudes of action potentials of the acoustic nerve in response to clicks were measured while the olivo-cochlear bundle was being stimulated, revealed that the stimulated tract produced an inhibitory effect upon acoustic nerve potentials. It is considered possible that the olivo-cochlear bundle through a cholinergic mechanism exerts an inhibitory or facilitory effect upon the hair cell acoustic nerve junction, depending upon the amount of acetylcholine released. Experiments are now in progress which, it is hoped, will clarify this matter.

SUMMARY.

1. Acetylcholinesterase was found in the organ of Corti of the cat.

2. The enzyme appeared to be within or near the nerve chalices for the hair cells rather than upon or in the hair cells themselves.

3. The row of inner hair cells showed a much greater indication of acetylcholinesterase activity than the outer hair cell rows.

4. Many nerve fibers oriented toward the organ of Corti also showed strong acetylcholinesterase activity.

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THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY CONVENTION CRUISE

The Virginia Society of Ophthalmology and Otolaryngology is sponsoring a convention cruise to Havana and Nassau on May 26 to June 2, 1956. Sailing from and returning to Norfolk, Virginia, the "Queen of Bermuda" will act as the hotel for the trip. Fare for seven days, $165.00 and up per person. Make reservations with United States Travel Agency, Inc., Washington, D. C.

THE EAR IN HEAD TRAUMA.[*][†]

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The temporal bone is frequently involved in craniocerebral injury. Since it houses the cochlear and vestibular mechanisms, as well as the VIIth cranial nerve, injuries in this neighborhood may cause serious sequelae. The object of this report is to present the mechanics involved in skull fractures and to review the otological disturbances resulting from head injuries. The subject is worthy of scrutiny because of the many new concepts which have been formed as a result of recent experimental and clinical studies on many of the problems bearing on the subject.

CLASSIFICATION AND MECHANISM OF SKULL FRACTURE.

One can usually predict in a general way the type of fracture that will result with a given intensity of force applied to a specific region of the skull. Variations are dependent upon the contour and consistency of the skull. This was determined by stresscoat and strain gauge studies.[1,2] This discussion will concern fractures involving the temporal bone.

The petrous portion of the temporal bone forms two-thirds of the floor of the middle fossa and one-third of the floor of the posterior fossa and, therefore is vulnerable in fractures involving the base of the skull. The foramina of the petrosa act as areas of stress concentration. Consequently, fracture lines in the vicinity may extend toward these foramina.

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The types of blows commonly associated with fractures of the temporal bone may be classified as follows:

1. Temporal and parietal area blows.

2. Occipital area blows.

3. Frontal area blows.

TEMPORAL AND PARIETAL AREA BLOWS.

These result in tearing apart forces of the base in an anteroposterior direction. A fracture produced by such a blow is vertically directed in the parietal and temporal bones, and may extend toward the base of the skull and top of the petrosa. Even though the petrosa is a buttress and may direct a fracture line anteriorly between it and the spheno-zygomatic buttress, yet when the stress is at right angles to the petrosa a fracture will frequently split or bisect it lengthwise, producing the commonly called longitudinal fracture. In severe cases the petrosa may become comminuted and fragmented with the fracture line continuing on into the foramen magnum (see Fig 10).

TYPES OF PARIETAL BLOWS:

1. Posteriorly to one side of the midline; frequently involves the bony external auditory canal (see Fig. 11).

2. Posterior edge of interparietal area at midline; (see Fig. 12). The fracture often extends into the mastoid process

3. Low, anterior parietal area; fracture line extends down and forward toward the anterior portion of the temporal fossa to involve the temporal squama and the greater wing of the sphenoid. The fracture may extend to the body of the sphenoid and even across to the other side (see Fig. 7).

Longitudinal fractures of the petrosa are usually extensions from fractures of the temporoparietal region passing down toward the external auditory meatus and then along the anterior aspect of the petrosa toward the Gasserian fossa.

OCCIPITAL AREA BLOWS.

These result in tearing apart forces of the base from side to side, particularly in the occipital squama (posterior fossa).

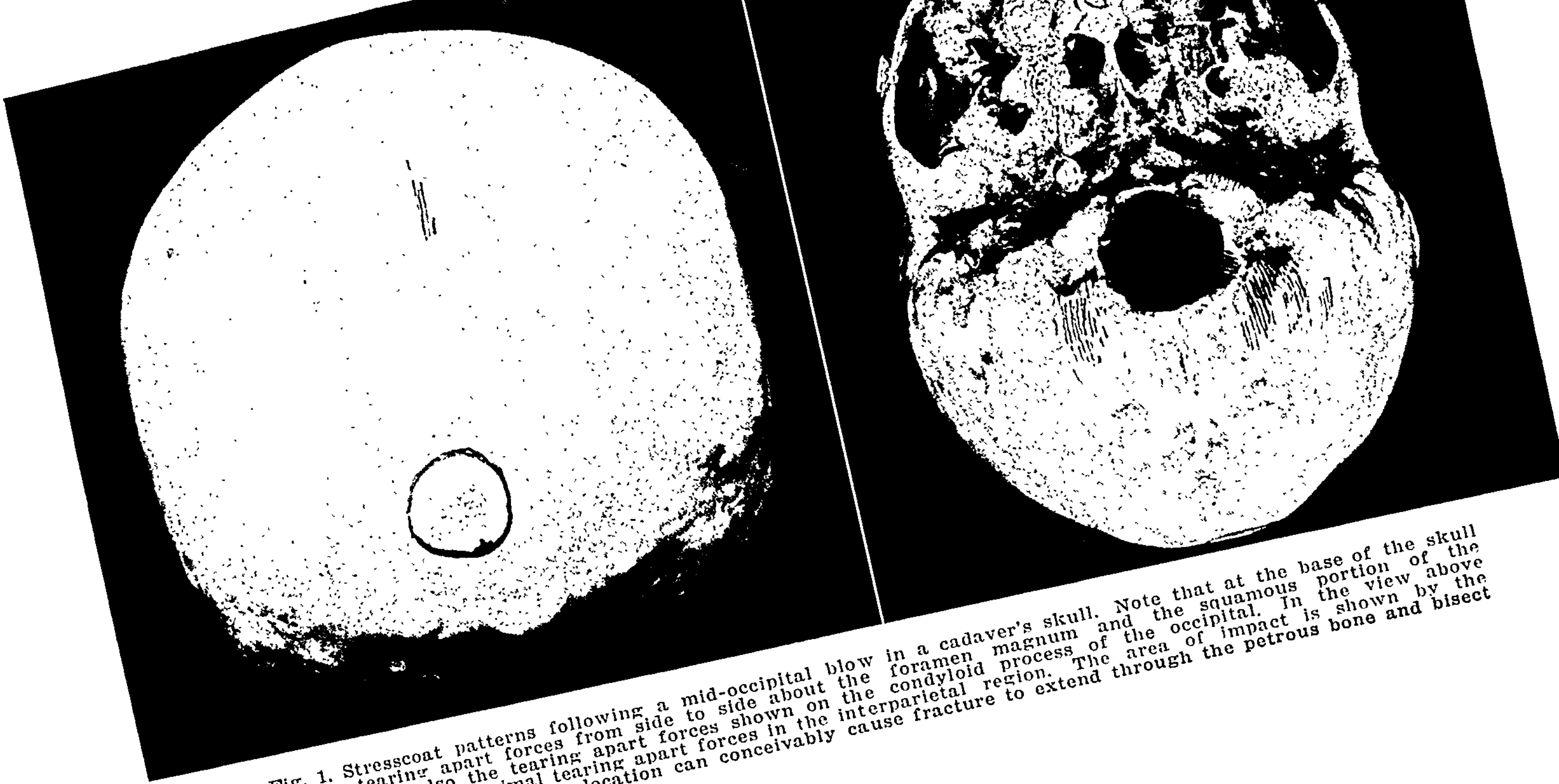

Fig. 1. Stresscoat patterns following a mid-occipital blow in a cadaver's skull. Note that at the base of the skull there are tearing apart forces from side to side about the foramen magnum and the squamous portion of the occipital. Note also the tearing apart forces shown on the condyloid process of the occipital. In the view above there is evidence of minimal tearing apart forces in the interparietal region. The area of impact is shown by the black circle. The blow in this location can conceivably cause fracture to extend through the petrous bone and bisect it on one or both sides transversely.

The fracture begins either in the vicinity of the foramen magnum or more laterally to involve the hypoglossal and jugular foramina. If the energy is adequate, the fracture extends forward through the basilar process of the sphenoid, fractures the petrosa transversely and ends in the middle fossa (see Figs. 1, 2, 3).

Transverse fractures of the petrosa are almost always

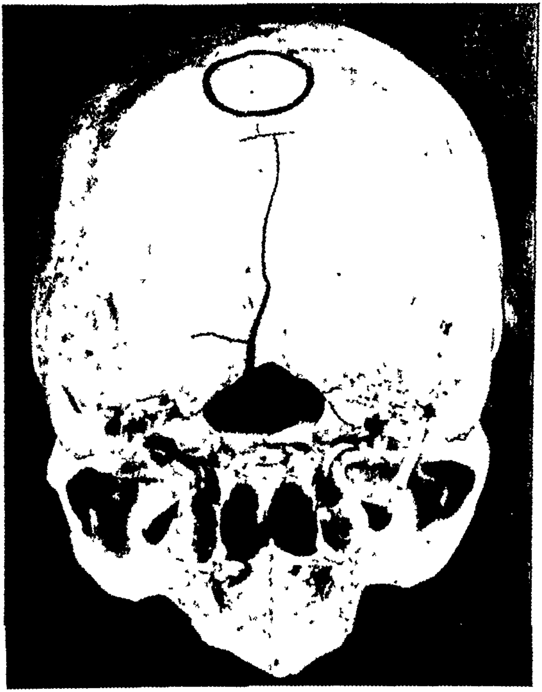

Fig. 2. Experimental skull fracture. Area of impact shown by black circle. The fracture line extending into the foramen magnum is much wider at the foramen than it is at the point of impact. Extensions of fracture toward the jugular foramen on both sides are noted. They also involve the condyloid process of the occipital.

extensions of posterior fossa fractures. They may, however, occasionally result from a posterior extension of an anterior fossa fracture (see Fig. 4).

In transverse fractures there is usually involvement of the internal acoustic meatus with severe injury to the VIIIth cranial nerve, resulting in a loss of cochleo-vestibular function. The VIIth nerve is often transected.

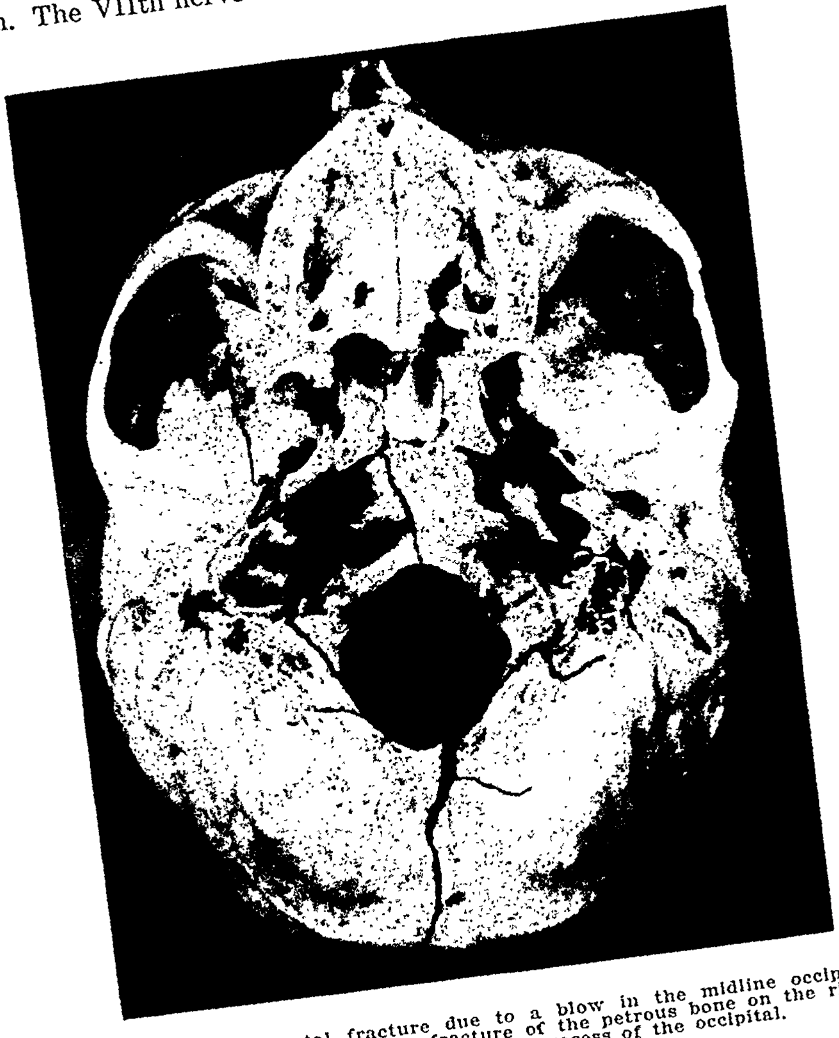

Fig. 3. Experimental fracture due to a blow in the midline occipital area resulting in a transverse fracture of the petrous bone on the right side as well as a fracture of the basilar process of the occipital.

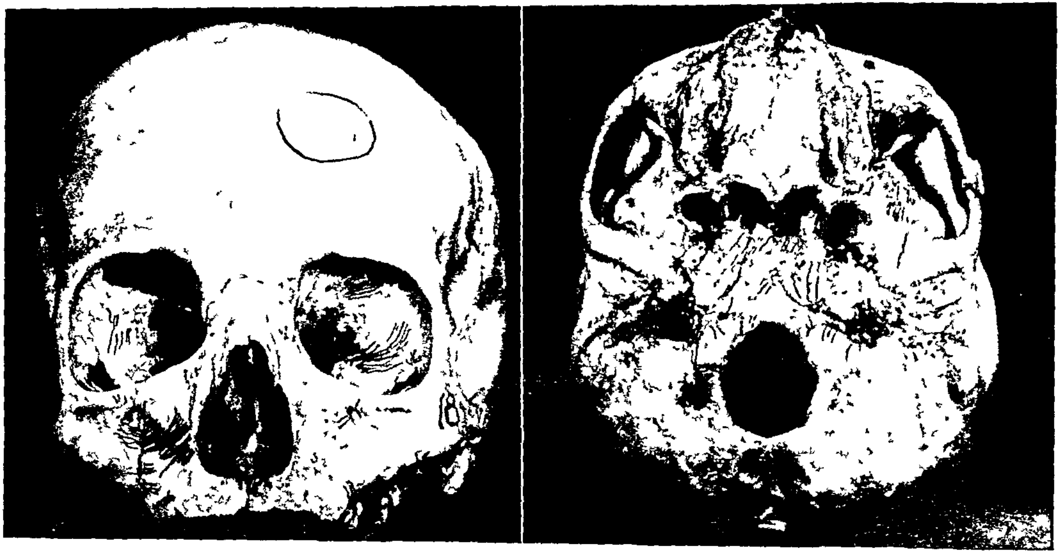

Fig 4 A left *frontal* blow in a cadaver's skull showing tearing apart *forces of* the orbits and the upper jaw as well as the base of the skull Note that with a blow of *sufficient* severity a fracture line may extend across the base of the skull obliquely from the left to the *right side* Such a fracture may involve the petrous bone on the same or on the opposite side and *in an oblique transverse* direction

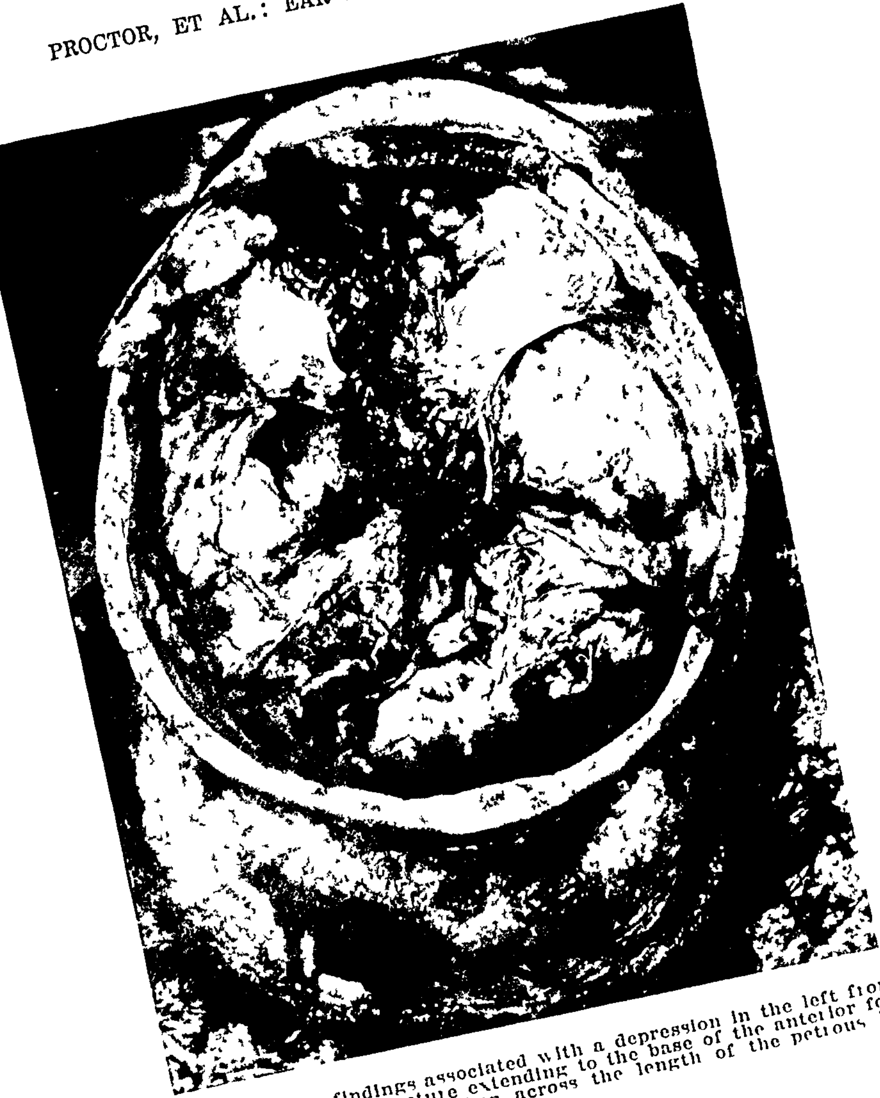

Fig 5 Autopsy findings associated with a depression in the left fronto-temporal area with a fracture extending to the base of the anterior fossa, to the pituitary region and then across the length of the petrous bone on the opposite side

Pure transverse or longitudinal fractures are uncommon. In either type comminution and fragmentation is usually present; furthermore, modifications and combinations of transverse and longitudinal fractures of the petrosa may occur. Combined longitudinal and transverse fractures may occur from severe upon the head, particularly in falls upon the vault from

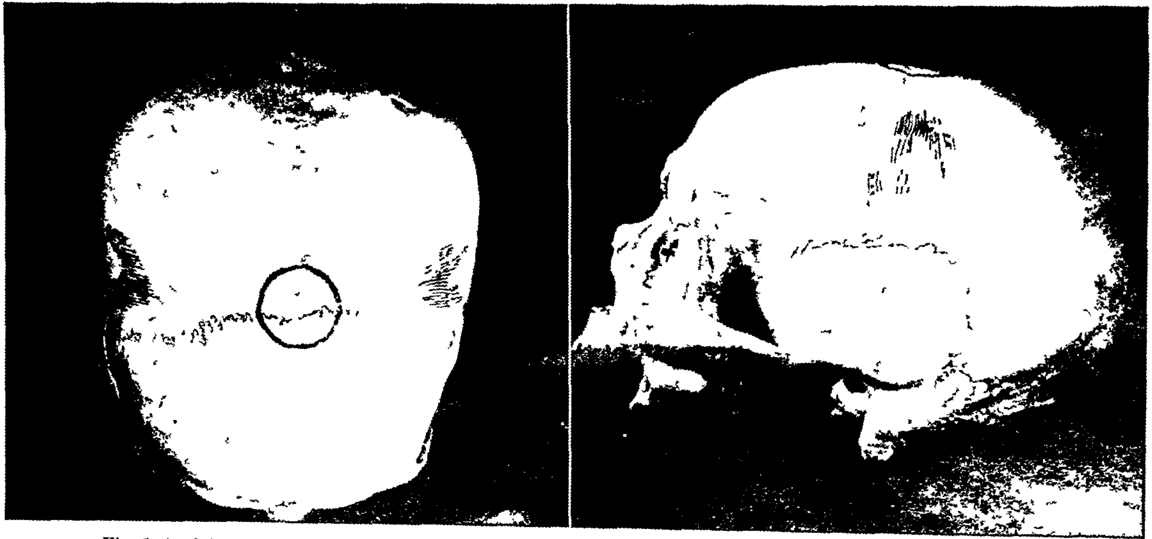

Fig [illegible] An interparietal blow at the frontal junction showing tearing apart forces involving the parietal bones on both sides The tearing apart forces are in an anteroposterior direction with the cracks in the lacquer extending superoinferiorly Such a blow may conceivably cause unilateral or bilateral fracture extending toward the mastoid and toward the external auditory canal Such a fracture can cause a longitudinal involvement of the petrous bone

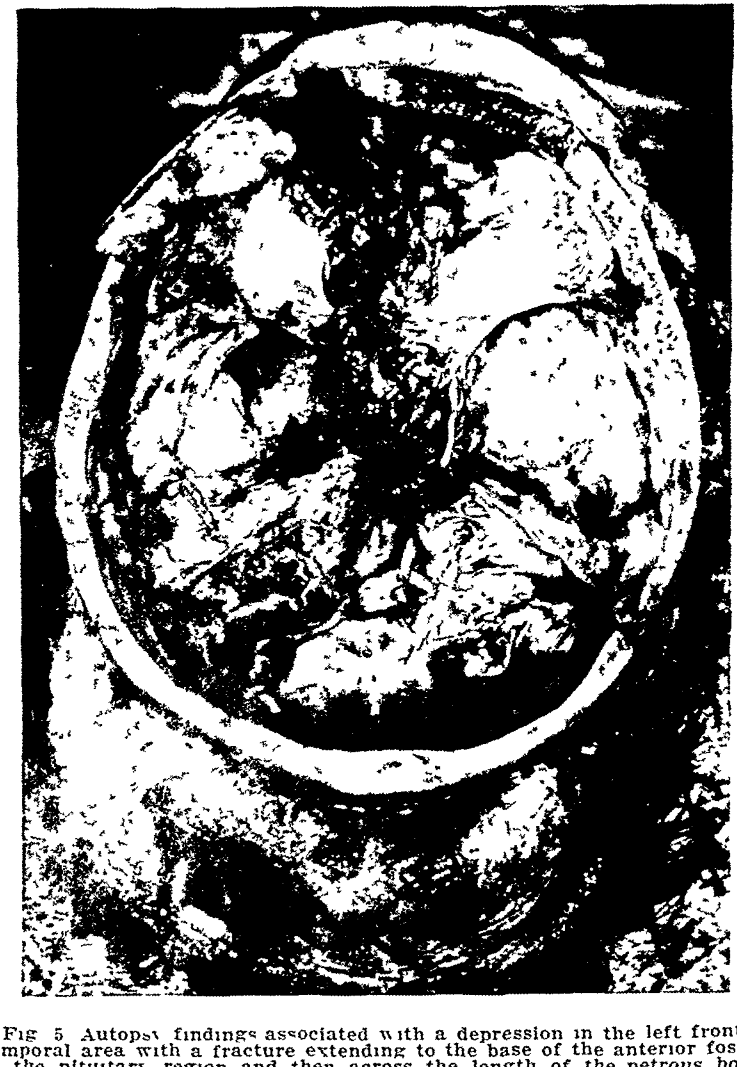

Fig 5 Autopsy findings associated with a depression in the left frontotemporal area with a fracture extending to the base of the anterior fossa, to the pituitary region and then across the length of the petrous bone on the opposite side

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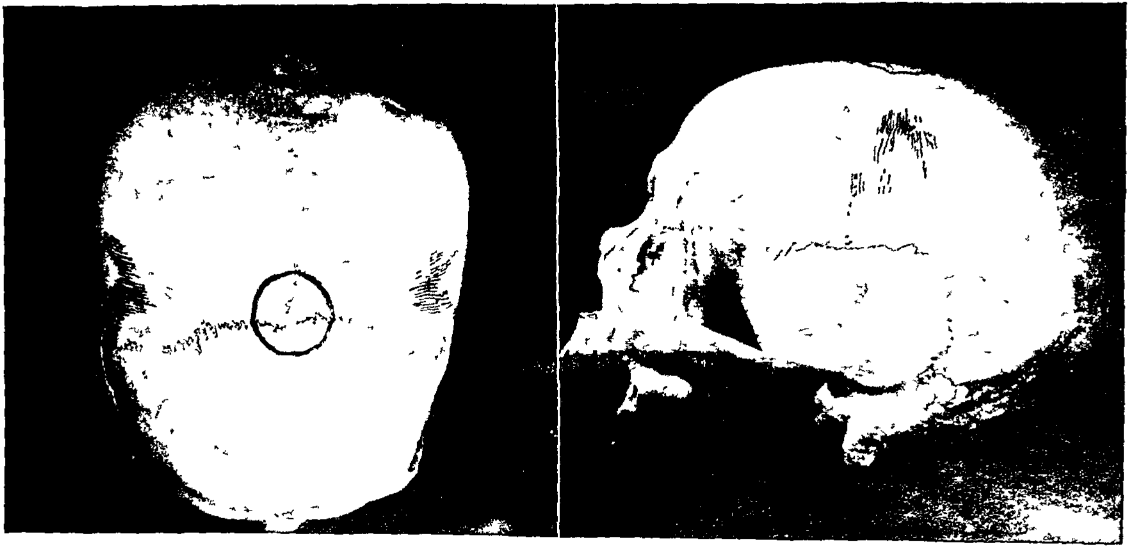

Fig 6 An interparietal blow at the frontal junction *showing tearing apart* forces involving the parietal bones on both sides *The tearing apart* forces are in an anteroposterior direction with the cracks in the lacquer extending superoinferiorly Such a blow may conceivably cause unilateral or bilateral fracture *extending toward the mastoid* and toward the external auditory *canal* Such a *fracture* can cause a longitudinal involvement of the petrous bone

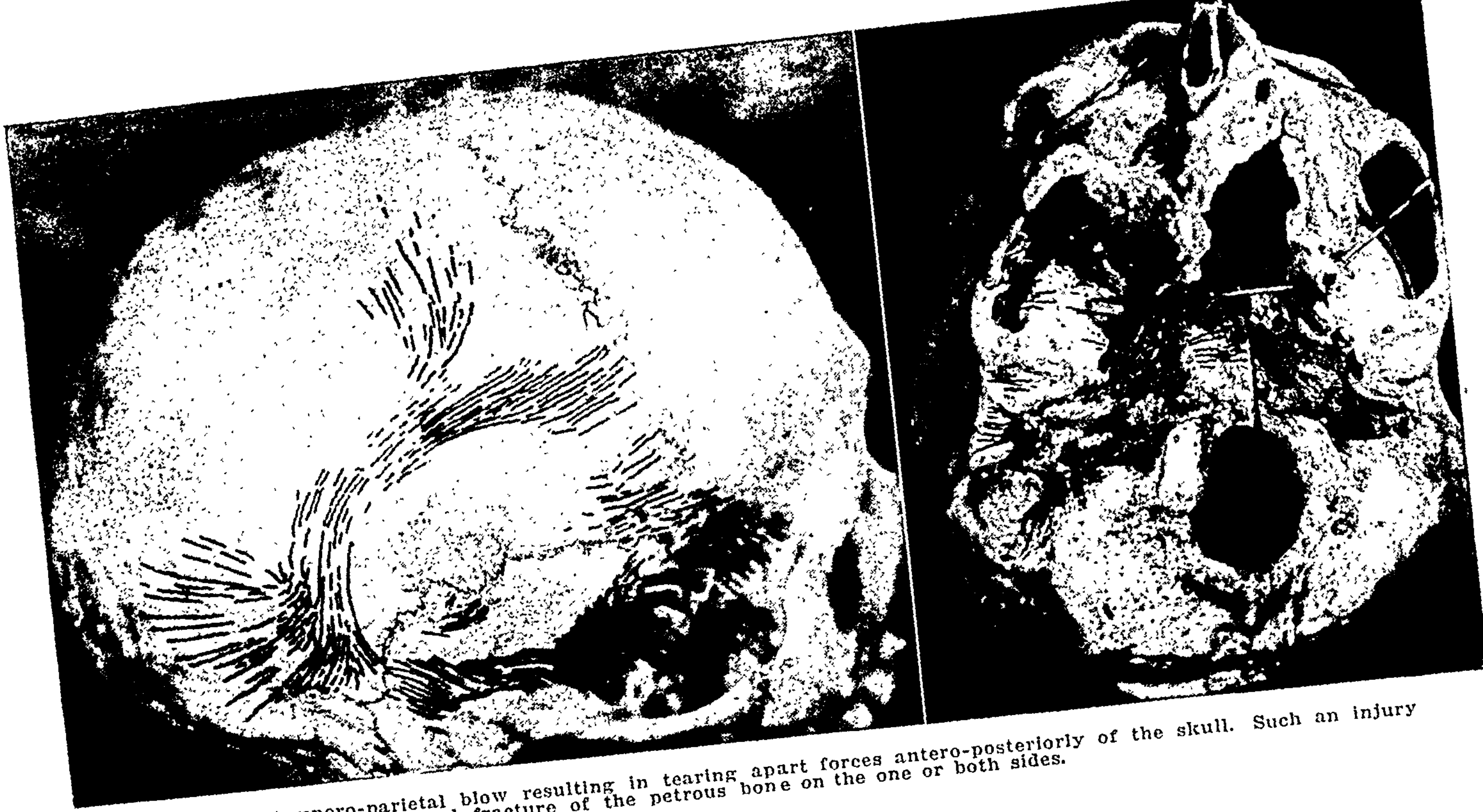

Fig. 7. A temporo-parietal blow resulting in tearing apart forces antero-posteriorly of the skull. Such an injury may result in a longitudinal fracture of the petrous bone on the one or both sides.

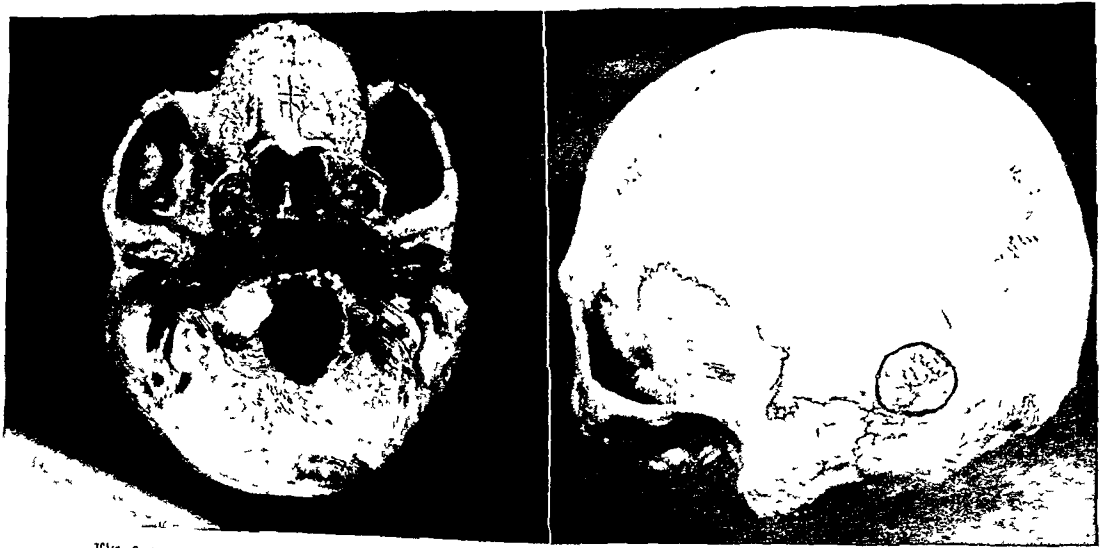

Fig 8 Impact above mastoid on left resulting in tearing apart forces on base slanting obliquely toward both petrous bones and the foramen magnum

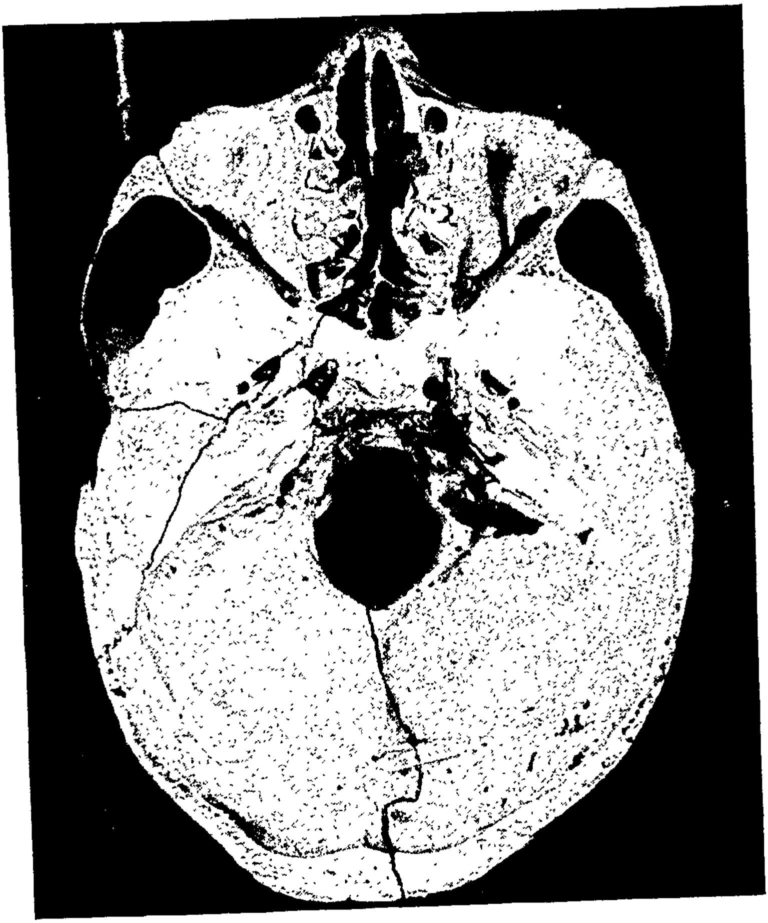

Fig. 9. Experimental fracture following a posterior vertex blow with lines of fracture extending to the foramen magnum in the midline posteriorly and toward the petrous bone, from the parietal down to the temporomastoid area. The fracture line involves the petrous bone longitudinally as seen in this figure. The petrous involvement is obliquely forward and medially.

a good height. Figure 10 illustrates a vault fracture extending toward the petrosa. It was caused by falling three floors and landing on the head. At autopsy the fracture was found to extend through the petrosa transversely and thence through the jugular and hypoglossal foramina to the foramen magnum, so that a triangular piece of bone was actually broken loose. The right XIIth nerve was paralyzed.

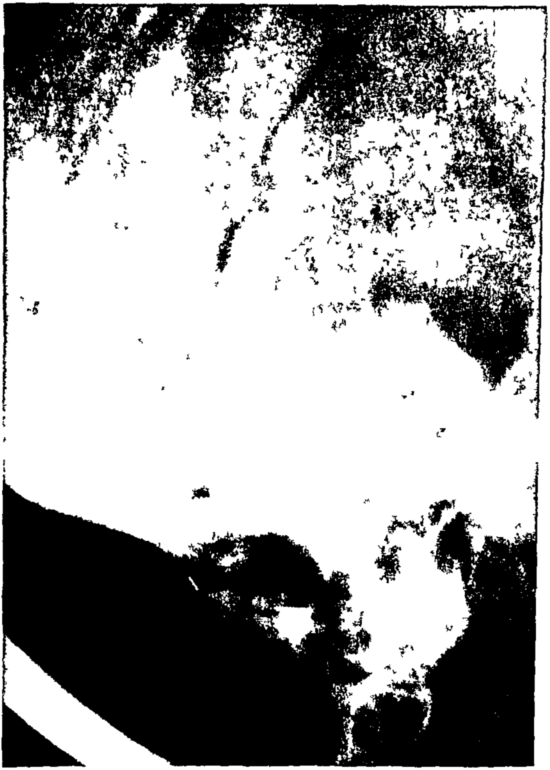

Fig 10 A parietal fracture extending down toward the mastoid area has involved the petrous bone longitudinally and the occipital bone surrounding the foramen magnum This patient had a twelfth nerve involvement before death Fracture extensions involving the condyloid foramen were noted at autopsy

FRONTAL BLOWS.

These result in tearing apart forces from side to side at the base of the skull in the anterior fossa. A frontal fracture involving the anterior fossa may extend into the middle fossa,

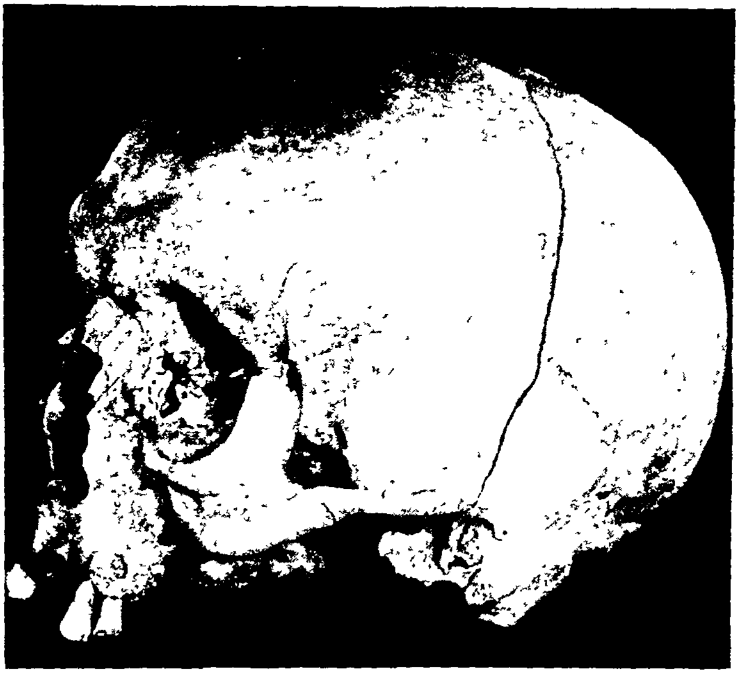

Fig. 11. Experimental skull fracture with impact at the vertex (circle). Note the involvement of the external auditory canal. Inside the skull such a fracture involves the length of the petrous pyramid ending in the region of the lateral pituitary area.

fracture the petrosa transversely and extend on into the posterior fossa (see Fig. 4). A lateral frontal blow (see Fig. 5), may result in an obliquely directed fracture of the base of the skull.

ISOLATED FRACTURES.

Isolated fractures of the base, unassociated with radiations into the vault, may involve the middle fossa and petrosa, but these are not common. In posterior parietal, interparietal and mid-occipital blows, such isolated stresses of the base were seen in about six per cent of the skulls studied. These fractures may be produced by:

1. Blows upon the vault.

2. Blows so applied as to cause an inbending about the foramen magnum from compression of the head against the neck or vice versa.

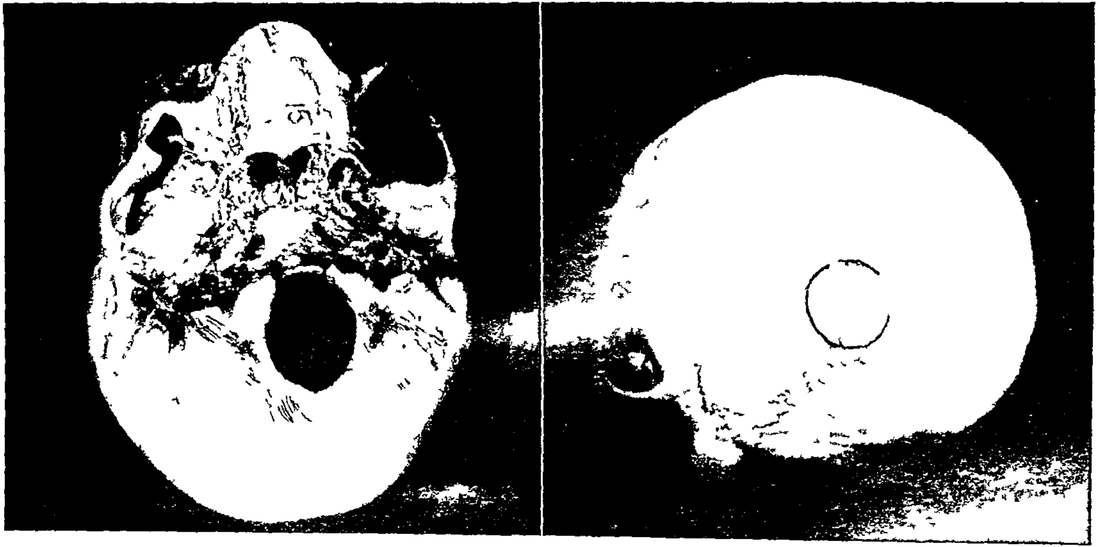

Fig 12 A left lateral parieto occipital blow near the mastoid resulting in tearing apart forces obliquely across the base of the skull in the occipital area extending toward the foramen magnum and toward the petrous bone on the opposite side

3. Blows applied to the temporo-mandibular joint.

4. Blows low along the circumference of the skull, particularly by sharp objects, may also involve the base primarily very much like a hatchet may tear through the grain of a piece of wood.[64]

PATHOLOGY.

Our knowledge of the pathological changes in the temporal bone as a result of head trauma, has been derived from autopsy studies (gross and microscopic examination of the temporal bone) and from animal experiments reported by various investigators.

The pathology in longitudinal fractures:[3,4,5]

1. The fracture courses through the roof of the middle ear and then along the anterior edge of the pyramid (through the superior wall of the Eustachian tube and carotid canal).

2. The tympanic membrane and external canal walls were often injured. The incus may be dislocated and the ossicular ligaments torn. The ossicle may be fractured.[4]

3. The labyrinthine capsule remains undamaged.

4. If the facial canal is involved, the damage usually occurs in the region of the genu. The facial nerve is usually spared.

5. The nerves to the internal ear are almost always injured by hemorrhage, tearing or stretching.

6. The vestibular end-organ is rarely damaged.

7. There is frequent evidence of injury to the membranous cochlea (hemorrhages into spiral ligament and scala tympani) especially near the round window.

8. Deafness is more of the conduction type. Occasionally inner ear deafness is pronounced and may be due to isolated cochlear fractures, direct damage to the hair cells by the traveling pressure wave, or damage to the higher centers.

The pathology in transverse fractures:[3]

1. The fracture courses at right angles to the pyramid.

2. The cochlea is more vulnerable than the vestibule.

3. The nerves in the internal auditory meatus may be torn.

4. There may occur a tearing of the membranous labyrinth.

5. Hemorrhages occur into endolymph as well as into perilymph.

6. The middle ear damage, if present, is confined to the mesial wall (musculo-tubal canal, knee of the facial, stapes footplate).

7. The facial nerve is often involved especially at the geniculate ganglion (in over 50 per cent of the cases). The paralysis may be permanent.

8. Hematotympanum occurs at times. External bleeding from the ear is rare.

9. Cerebrospinal otorrhea is more common in transverse than in longitudinal fracture. This fluid may drain down the Eustachian tube.

The pathology of isolated labyrinthine fractures has been reviewed by Grove.[6] Usually if the labyrinthine capsule is fractured, a complete loss of cochlea-vestibular function ensues. Occasionally, however, an isolated fracture of either cochlea or vestibule may occur with an isolated loss of function of one portion and a partial to complete preservation of function of the other portion of the labyrinth.

Studies of inner ear changes after mild injuries were made in animals by Stenger.[7] Following light blows with a hammer on the skulls of white rats, he found hemorrhages in the region of the round windows and in the basal coil of the cochlea. The vestibule showed no changes. With heavier blows the hemorrhages were more severe at the round window and extended to the helicotrema. Some bleeding was noted in the cochlear nerve and in the ampullae. Similar experiments were conducted by Brunner[8] on guinea pigs, and they confirmed Stenger's findings.

Pathological changes found in the temporal bones of those dying soon after injury: the changes found microscopically in animal experiments and the changes found in the temporal bones of those who died many years after head trauma, are quite similar. Grove[6] states that these changes consist of stretching and tearing of nerves to various degrees, hemorrhages in various locations but particularly in the perilymphatic spaces of the basal coil of the cochlea in the vicinity of the round window. "The exudation of blood is probably partly absorbed and partly organized, leading to atrophy of the finer nerve branches and degeneration of the neuroepithelium of both cochlea and vestibule."

In the event of extensive bleeding into the labyrinth, there is a strong likelihood of extensive connective tissue organization of the blood clots. Later, this reparative connective tissue invasion may in turn be replaced by new bone formation within the confines of the osseous labyrinth.[3,9]

Labyrinthine fractures heal with new bone formation in the endosteal and periosteal layers. The enchondral layer heals only with connective tissue.[10] The healing is fairly well advanced in one month. Occasionally these fractures appear to heal incompletely or not at all, so that subsequent middle ear infection might well diffuse through these old fracture lines and reach the meninges. These unhealed fractures may be discernible in Roentgen studies many years later.

Many cases of head trauma, without clinical or radiological evidence of skull fracture, develop deafness and vestibular disturbances. This may be due to hemorrhages into the labyrinth[4,7,8,11] or to severe degenerative changes in Corti's organ[12,13,14] or to pulling and tearing of the cochlear nerve in the internal auditory meatus.[3,15,16]

The term "commotio labyrinthi" has been used to describe the presence of a hemorrhage into the inner ear. Grove[6] considered labyrinthine concussion or commotio as the commoner cause of deafness and vestibular disturbances. He pointed out that hearing loss with a fractured skull and contralateral hearing loss in fracture cases can be explained on the basis of a commotio or inner ear hemorrhage. Ballance[17] has used the term "concussion" to describe this state. Ballance believed that hearing loss may result not only from injury to the peripheral mechanisms but also from damage to the central auditory pathways by the commotion.

Oppenheim suggested that commotio cerebri may lead to hearing loss by injury to the cochlear nuclei or higher centers. Because of the partial crossing of the ascending acoustic fiber systems, central lesions will give few if any symptoms[17]. Lesions of the medial geniculate body or the auditory cortex will not result in deafness unless bilateral, according to Tobey.[18]

Normal auditory thresholds in the cat were found by Neff after bilateral destruction of the auditory cortex.[19] Schuknecht and Woellner have shown that up to 75 per cent of nerve fibers supplying a particular region of the cochlea can be destroyed without creating threshold losses for frequencies having their locus in that region.[20,21] The clinico-pathological evidence of true central deafness is inconclusive. Fraser and Rowe[22] reported a reduction in hearing in some instances of temporal lobe tumors. Unilateral removal of the temporal lobe has not influenced hearing, according to Penfield and Erickson.[23]

Moderate blows with a hammer were delivered to the exposed mastoid of human volunteers by Ruedi and Furrer[24] and by Escher.[25,26] The subjects described an acoustic sensation resembling a pistol shot. Audiograms showed mild 4,000 cycle dips which disappeared after 24 to 48 hours. Lindsay stated that the blows of a mallet at mastoidectomy may cause permanent hearing loss at 4,000 cycles.[27]

Schuknecht, Neff, Perlman[28] subjected conditioned cats to head blows. Audiograms were taken before and after the trauma. They were able to demonstrate 15 to 40 db recovery of hearing acuity over the frequency range tested during the first two weeks following the head blow. They also showed that deafness was more easily produced when the blows were delivered to the skull near the experimental ear. Gross and microscopic studies of the temporal bone and brain failed to reveal significant pathologic lesions in the middle ear and brain. With graphic reconstructions of the cochlea they found the primary injury to be a degenerative change in the organ of Corti with the upper part of the basal coil most susceptible to injury. They found, in the order of severity, these recognizable stages of end organ damage:

1. Loss of external hair cells.
2. Loss of external and internal hair cells.
3. Flattening of the organ of Corti.
4. Complete disappearance of the organ of Corti.

There was secondary degeneration of the nerve fibers and ganglion cells supplying the damaged regions of the cochlea. Perlman found that maximum nerve degeneration occurs three weeks after the head injury.[10]

According to Schuknecht[29] blows to the head create pressure waves in the bones of the skull which are transmitted through bone to the cochlea, where they produce a "shock pulse" in the fluids of the inner ear. This phenomenon is comparable to the effect of an airborne blast wave or intense noise on the tympanic membrane which is transmitted by the conductive mechanism to the inner ear fluids. These pressure waves displace the basilar membrane during analysis of the acoustic stimulation. Intense stimulation produces a violent displacement of the basilar membrane causing injury to the organ of Corti. Both reversible and irreversible cellular injury occurs in this end organ.

Schuknecht further observed that hemorrhage into the labyrinth did not appear to be responsible for deafness. "The location of blood had no correlation with the location of end-organ damage. In view of the findings it appears unlikely that blood cells act as an irritant in an intact labyrinth. Blood was found in the cochlea as long as four months after the head blow without causing reaction within the labyrinth."

INCIDENCE OF EAR INVOLVEMENT IN CRANIOCEREBRAL INJURY.

A series of 152 consecutive head injuries were examined by Grove[6] within 24 hours. There was evidence of skull fracture in 17 cases and ear signs or symptoms in 49 cases (32.66 per cent). Mygind[3] in otoscopic examination of similar cases found traumatic changes in about 50 per cent. In one-half, however, only a hyperemia of the drum was present. Mygind found evidence of vestibular trauma in one-third of the cases; Grove found an incidence of 26.6 per cent. Gurdjian[31] reported a series of 718 acute cranial injuries seen during an 18-month period. Of these, 475 were proven to have skull fracture

(X-ray, inspection and autopsy). Bleeding from the ears occurred in 129 cases. Clinical evidence of brain damage was found in 246 cases. Alexander and Scholl[32] reported demonstrable hearing losses in one-third of their reported series of 551 cases of head trauma.

In our present study of 100 consecutive cases of head trauma seen for otological evaluation, 98 revealed signs and symptoms referable to disturbances of structures contained within the temporal bone. It is our firm belief that the great majority of cranio-cerebral injuries will present subjective (tinnitus, vertigo, deafness, bleeding, headaches) or objective (unconsciousness, audiometric hearing loss, abnormal caloric test, spontaneous nystagmus, positional nystagmus, deformities of the external canal and drum, X-ray evidence of fracture, facial nerve paresis or paralysis) evidence of fracture or concussion.

OTOSCOPIC EXAMINATION.

A careful examination of the external canal and drum should be done as soon after the injury as possible. Lacerations in the skin may heal rapidly. Hematotympanum and liquor tympanum may be detected for only a few days. Fracture of the bony canal wall may be diagnosed by the presence of blood clots, or bloody crusts on its walls, or in the skin of the canal (ecchymoses), and by irregularities in the bony configuration of the canal. Deformities in the external canal wall were detected in eight of 28 longitudinal fractures in our series. The external canal is never fractured in pure transverse fractures.

BLEEDING FROM THE EAR.

Following a head injury bleeding may occur from the ear simply as a result of a laceration within the external auditory canal or from rupture of the tympanic membrane. In either event the amount of bleeding is minimal. If the hemorrhage is profuse it may be the result of injury to a large vein (lateral or superior petrosal sinus, jugular bulb), tympanic plexus of veins and arteries or the middle meningeal artery. Rarely is the carotid artery involved. If it is, there is profuse bleeding from the nose, mouth and ear. At times in spite of an absence of a history of bleeding from the ear the examiner observes blood clots, ecchymosis or a hematoma on the walls of the external auditory canal, occurring most often on the posterior and superior walls of the canal.

Another sign of temporal bone fracture is the development of an ecchymosis over the mastoid area (Battle). This usually appears on the fourth or fifth day after injury.

Active bleeding in our experience usually stops spontaneously when the patient is placed in the upright position. It has never been necessary to expose the source of bleeding in patients with hemorrhage from the external auditory canal.

In transverse fractures bleeding into the middle ear is frequent without rupture of the tympanic membrane. This is discernible on otoscopic examination, and is called hematotympanum. The drum has a blue discoloration and bulges. Grove found this sign in seven cases. We observed this sign in two cases out of a hundred.

In Grove's 211 cases of skull fracture 112 bled from one ear and 34 from both ears. In 530 cases of skull fracture reported by Gurdjian[33] unilateral hemorrhage occurred in 281 and bilateral hemorrhage in 47 cases. In our study, which included 57 patients with skull fractures, bleeding occurred from the ear in 24 cases. Davis[34] reported a mortality of about 66 per cent in patients with bilateral hemorrhage, while in unilateral hemorrhage the mortality was 31 per cent. Bleeding from the ear is reported to occur in about 30 per cent of head injuries (24 per cent in our study).

Delayed or late hemorrhage from one ear may occur, and several such cases have been reported by Grove.[35] In two cases the hemorrhage occurred one month after the injury. This may have been due to involvement of a small vessel in an area of osteomyelitis or osteitis with erosion and delayed bleeding.

Fracture of the skull is frequently present in association with bleeding from the ear. Gurdjian reported 87 per cent demonstrable fractures in 129 cases of bleeding from the ear.[31] Among the 84 cases with unilateral bleeding in this group there were 73 with positive Roentgen evidence of fracture and 11 with negative findings. In this same group the site of the fracture was in the middle fossa in 48 cases, in the posterior fossa in 29 cases, and in the anterior fossa in 15 cases. In a recent study of Gurdjian and Webster[16] of 1285 cases of head injury there were 184 cases with bleeding from the ear and six cases with cerebrospinal otorrhea. Of the 184 cases with a bloody otorrhea 164 had a skull fracture —88 per cent. The mortality rate of those with bleeding from the ear was 17½ per cent, a figure much lower than in earlier reports, probably due to antibiotics and sulfonamides.

CEREBROSPINAL OTORRHEA.

The incidence of cerebrospinal fluid otorrhea has been reported by several authors. Besley[37] reported it in 20 patients among 1,000 with skull fractures; Yerger,[38] in 20 per cent of skull fractures; Grove,[6] in eight of 211 cases. We found it in two of 57 cases with skull fracture. The series of 718 head trauma cases with 475 proven skull fractures reported by Gurdjian[31] presented cerebrospinal fluid otorrhea in eight out of 129 cases with bleeding from one or both ears.

The otorrhea may be difficult to identify for several days because of the presence of blood; furthermore, the cerebrospinal fluid otorrhea may be scanty and of short duration so that it is not detected. On the other hand it may be copious (Ballance[17] reported a case which yielded 600 cc. of fluid within 24 hours) and may persist for several weeks.

Grove[6] felt that cerebrospinal fluid otorrhea is an indication of a longitudinal fracture of the temporal bone. Occasionally the onset of the cerebrospinal fluid otorrhea is delayed [6,39,40]. This type of otorrhea has been observed behind an unruptured drum.[41,42] This may be identified in an otoscopic examination by a clear fluid level in the tympanum.

Cerebrospinal fluid otorrhea usually subsides spontaneously and rarely requires surgical treatment. When it is accompanied by discharge of brain tissue, surgical treatment is indicated The management is similar to that of an open skull fracture. We have had five instances of cerebrospinal fluid otorrhea and brain discharge from the ear during the past ten years. In such cases debridement with closure of the dural tear is performed. If the dural tear is large a piece of temporal fascia or subcutaneous fascia from the scalp may be used for closure.

UNCONSCIOUSNESS.

In general, the more severe the injury the greater the likelihood of unconsciousness and the longer its duration. In Grove's series of 211 skull fractures involving the temporal bone there were 28 cases without unconsciousness. In our study of 100 cases of head trauma we found the following incidence of unconsciousness:

1. Transverse fractures, 16—unconsciousness, 9.

2. Longitudinal fractures, 28—unconsciousness, 9.

3. Skull fractures without temporal bone fracture, 13—unconsciousness, 3.

4. Head injury without fracture but with unconsciousness, 14.

5. Head injury without fracture or unconsciousness, 32.

TINNITUS.

Tinnitus is a common complaint after head trauma. It is not necessarily in proportion to the degree of trauma or of deafness and occasionally remains as the only residual disturbance.

In the 100 cases of *head trauma* in this study we found six of 16 cases of transverse fracture, seven of 28 cases of longitudinal fracture, three of 13 cases of skull fracture not involving the temporal bone, six of 14 cases of head injury without fracture but with unconsciousness, and 14 of 32 cases of head injury without fracture or unconsciousness, to have tinnitus.

DEAFNESS.

Head trauma frequently results in some degree of deafness. This may occur without the presence of a skull fracture, in which case it is commonly referred to as concussion deafness. A moderate blow to the head, which does not produce a fracture, will often produce a hearing loss varying from a mild dip at 4,000 cycles to severe inner ear deafness. The losses are most severe for the tones between 3,000 and 8,000 cycles. In the severe cases the entire audible frequency range is usually involved with the greatest loss at the higher frequencies.

The hearing function is determined with the audiometer. Supplementary testing is done with the recorded voice tests and tuning forks. It is important to delay the testing until the sensorium is clear. Since both conductive and perceptive structures in the hearing mechanism are frequently affected by head trauma the audiogram taken shortly after the head injury may show a uniform hearing loss for all frequencies. Conductive lesions tend to resolve whereas perceptive lesions frequently do not or only partially recover so that the degree of hearing impairment will vary.

Audiometric studies on recovery have been reported by Schuknecht.[43] Usually deafness is noted immediately after the injury, but it may not become manifest until later, when secondary degenerative changes in the cochlea take place producing a progressive deterioration of hearing. Loudness recruitment is present in perceptive deafness resulting from head injury. Since recruitment is absent in cochlear nerve lesions and present in Meniere's disease and noise deafness it strongly supports the concept that head trauma damages the organ of Corti.[21]

The tests for hearing are subjective, consequently one must be constantly aware of the possibility of malingering. Another important consideration is that there is usually no knowledge of the patient's hearing function before the time of injury. This is an important factor in medico-legal cases which involve settlements.

Saltzman[44] believes that head trauma may damage the cochlear nuclei and secondary auditory tracts, resulting in a loss of low and high tones by air conduction. The bone conduction curve varies from frequency to frequency and does not follow the trend of the air conduction curve. He stated that this pattern appears several weeks after the head injury. We have attempted to evaluate the hearing loss in the following groups of head trauma cases:

1. Longitudinal fracture of the temporal bone.
2. Transverse fracture of the temporal bone.
3. Skull fracture without temporal bone fracture.
4. Head trauma with unconsciousness but without evidence of fracture.
5. Head trauma without unconsciousness or fracture.

Longitudinal Fracture of the Temporal Bone—The hearing was examined in 28 cases of longitudinal fracture. Three of these also had a transverse fracture, leaving a total of 25 cases with only longitudinal fractures. Of these nine had variable degrees of nerve deafness; 13 had mixed conduction and nerve deafness, and three cases demonstrated no hearing loss one to six weeks after the accident. Schuknecht[43] states that "the occurrence of conduction deafness alone in the fractured ear is uncommon. Some degree of inner ear deafness is almost always associated with the conduction loss." We found, too, as reported by Ruttin[45] and by Schuknecht[43] that when a hearing loss is demonstrated in the ear opposite the fracture it is of the inner ear type (concussion deafness).

The prognosis for recovery of hearing in longitudinal fractures is usually favorable. The conduction loss due to middle ear bleeding and trauma usually recovers. The perceptive loss in hearing in these cases has a poorer prognosis with a much less degree of recovery.[3,6,8,43]

Grove[6] stated that the function of hearing is more severely damaged in longitudinal fracture with increases in age; furthermore he tested the hearing in 108 of 112 cases of longitudinal fracture, and found loss of hearing in 79.5 per cent on the homolateral and 54.6 per cent on the contralateral side, indicating that labyrinthine concussion is of common occurrence in head trauma.

According to Skoog[16] longitudinal fractures present either a conduction deafness (65 per cent), perceptive deafness (22 per cent), or a mixed deafness (13 per cent).

Transverse Fracture of the Temporal Bone—In our study we had 16 cases all with total deafness on the affected side. On the contralateral side eight cases demonstrated a perceptive type of hearing loss. Grove[6] reported 16 cases of transverse fracture, all with homolateral complete deafness. He reported considerable damage to the contralateral ear in 65 per cent of these cases. Of interest are the cases reported by Kingenberg,[16] and Nager[9] in which fracture lines were visualized extending through the pyramid and in which vestibular function was intact but the cochlear function lost.

Skull Fracture Without Temporal Bone Fracture—A total of 13 such cases was examined. A bilateral nerve type hearing loss of variable degree was present in six cases. Two cases presented a unilateral nerve deafness, one of which was severe. Grove[6] reported 49 such cases. In testing the 98 ears of these cases he found 45 with indication of inner ear damage. Even after eliminating malingerers and those with a hearing loss before injury, Grove felt that in many of these ears the loss in hearing was due to injury either from stretching of the cochlear nerve (side motion of the brain according to Ulrich[3]) or from hemorrhage into the inner ear. We believe that the hearing loss is due to the shock pulse injury to the organ of Corti as described by Schuknecht.[43]

Head-Trauma With Unconsciousness but Without Evidence of Fracture—A total of 14 cases were examined in this group. Four had normal hearing; eight had a high tone loss and two presented a low tone loss. The group is small, but it suggests a rather high incidence of inner ear damage even though there is no clinical evidence of skull fracture.

Head Trauma Without Fracture or Unconsciousness—A total of 32 cases were examined. Normal audiograms were obtained in seven cases. A mild degree of bilateral nerve deafness was present in 12 cases. In five cases a severe degree of bilateral nerve deafness was present in two of which there was a previous history of acoustic trauma, and in one case central nervous system syphilis was uncovered. Four cases presented a unilateral total loss of hearing. Two cases had a previous conduction deafness due to chronic middle ear disease.

DISTURBANCES OF EQUILIBRIUM.

True vertigo and dizziness are common sequelae of head injury. These symptoms may arise from disturbances in the vestibular sense organs, the primary vestibular nuclei and probably the central vestibular pathway.[47] In a subconcussion, vestibular disturbances may be absent, but in a severe concussion these disturbances are common.

Two types of vestibular disturbances may occur as a result of head trauma: one is the peripheral type, the result of derangement of the vestibular end-organ; the other, the central type due to derangement of the primary vestibular nuclei and central vestibular pathways.

The peripheral type usually presents a true turning vertigo (staggering, room movements) in short attacks. Spontaneous nystagmus, with the quick component to the opposite side, and disturbance in balance and past-pointing may be noted. Accompanying involvement of the autonomic system may be present with nausea and vomiting; perspiration with a cold, clammy skin; a rapid pulse and lowered blood pressure. These effects are probably mediated through connections of the vestibular nuclei with the vagus. Asymmetrical caloric response may be present. This type of vertigo usually subsides in several weeks. The transitory nature of the symptoms has been explained by the ability of the vestibular nuclei to compensate for the unbalanced impulses which one deranged labyrinth may initiate.

McNally[48] has indicated that dizziness which arises from a disturbance in the central nervous system may be difficult to differentiate from an attack of peripheral labyrinthine dizziness. If tinnitus and hearing loss are combined with true vertigo in short attacks and autonomic disturbances are present, the likelihood of a peripheral origin is more certain.

Errors of sensation (postural vertigo) such as tilting of the bed, a rising of the floor, a falling of the ceiling of a room, a feeling of uncertainty or of being drunk, were believed by Grove[6,49] to be of possible vestibular origin if they were accompanied by an abnormal caloric response. Postural vertigo may be due to derangement in the utricular macula as a result of the pressure pulse transmitted to the vestibule as a result of a head blow. The utricular macula presents a gelatinous mass on its surface which contains calcium carbonate crystals. This mass (otolithic membrane) may be displaced by the forces of linear acceleration and gravity. A head blow can conceivably injure this delicate mechanism.[21]

The central type of vestibular disturbance is usually a sensation of disequilibration and not a sensation of true rotation. It is a common accompaniment of head injury. The term "blacking out" is sometimes used by patients to describe the attacks which may be initiated by sudden movements, such as stooping, straining, head turning and upward movement of the eyes. Rowbotham[50] attributes these momentary blackouts to instability of the cerebral circulation consequent upon injury to its vasomotor apparatus, rather than to labyrinthine dysfunction. Thus, he believes, a momentary ischemia of the brain, due to circulatory lability, is the essential cause of the dizziness. The usual disequilibration seen in patients with head injury is more suggestive of brain stem involvement with implication of not only vestibular fibers but also of tecto-spinal, rubro-spinal and spino-cerebellar pathways.

The peripheral vestibular elements may be injured directly by the pressure pulse set up in the labyrinthian fluids by the head blow. Such cases are known to have hemorrhages into the inner ear fluids which may be a cause for disturbed vestibular function.[27] A more important factor may be injury to the nuclei and central pathways in the brain stem.

Recent work of the mechanism of concussion reveals that in closed head wounds, pressure gradients at the cranio-spinal junction result in shear and tear of the elements in the brain stem. Even subconcussive blows in dogs resulted in some cellular changes of the medulla, pons and midbrain. These reversible changes (subconcussive blows) may occur in the cells of the brain stem explaining the usual findings in the human case of concussion.[51] Histologic changes have been demonstrated in the vestibular nuclei of animals after concussion injuries.[52]

Concussion is defined as a head trauma with unconsciousness at impact due to involvement of the brain stem reticular formation, associated with a shock-like state and slow pulse. Subconcussion is a head trauma not severe enough to cause post-traumatic unconsciousness but nevertheless associated with some involvement of the reticular formation.

The vertigo which follows cerebral concussion without deafness appears to be explainable by the alterations in the central vestibular nuclei.[47,52,53] That involvement of the brain stem pathways is important in the development of dizziness is suggested by the relative absence of dizziness in open wounds of the head where dissipation of injury at and about the open wound results in little or no involvement of the brain stem centers.

That disequilibration may be indicative of a derangement in the central nervous system is suggested by Leidler[54] who lists the following symptoms as strongly suggestive of a central lesion:

1. Normal hearing combined with either attacks of vertigo or hyper-excitability of one labyrinth or spontaneous nystagmus; *2.* Headache and vertigo; *3.* Positional nystagmus; *4.* Spontaneous nystagmus without vertigo; *5.* Preponderance of the slow phase of the nystagmus; and *6.* Rhythmic disturbances in vestibular reactions.

The following symptoms were listed by Van Egmond, Groen, Hulk and Jongkees[55] as proof of a central origin:

1. Spontaneous nystagmus towards a dead labyrinth or spontaneous nystagmus without diminution during a week; *2.* Purely rotational nystagmus of long duration; *3.* Vertical nystagmus; *4.* Normal turning reactions without caloric excitability; *5.* Monophasic reactions without quick phase; and *6.* Somnolence in deviations of the positions of the head.

The dizziness consequent to head trauma is usually positional in character and varies in severity. Positional nystagmus is a spontaneous nystagmus which is not constantly present but develops when the head assumes a particular position in space.[56] The various positions used in the test have to be assumed rapidly in many instances of head trauma before the nystagmus is elicited. Like the caloric test, responses to positional testing are likely to vary on repeated examinations in these cases of head trauma. The positional tests may have to be repeated a number of times before the symptom is produced and the nystagmus seen.

In head trauma cases demonstrating a positional nystagmus and, especially when the hearing is not impaired, a positional nystagmus of variable direction is seen (Type I of Nylen and Lindsay).[57,58,59,60]

Murphy[61] found 32 cases of positional nystagmus in 176 cases of head trauma. He found that the results varied on repeated examinations. He felt that this test is valuable when positive, since it is never positive in normal persons and left no question of normal limits to be considered.

The direction-changing nystagmus frequently elicited in head trauma on postural testing suggests a disturbance in the vicinity of the vestibular nuclei.[60] According to Lindsay the vestibular centers may be considered as normally in a state of equilibrium. When a lesion occurs in the vicinity of the vestibular nuclei the ability to compensate for positional changes may be impaired.

Cupulometry has recently been reported to offer quantitative data on the function of the vestibule with respect to nystagmus and the sensation of vertigo.[58] This is a turning test with small regulable stimuli of a magnitude provoked by ordinary life movements. The subject is rotated at specified speeds of turning, and the duration of the resulting nystagmus is noted. At the same time the subjective duration of the sensation of vertigo is recorded. The duration of nystagmus and sensation of turning are plotted on a logarithmic scale. The resulting graph is the cupulogram. According to Jongkees, in many cases cupulometry has been the only examination which made the clinical picture clear, or gave an objective basis for subjective complaints.[62]

The nystagmus and vertigo seen in the acute stage of a total loss of unilateral vestibular function (transverse fracture) are severe. The direction of the horizontal nystagmus is toward the side of the normal ear, the subjective vertigo in the direction of the nystagmus and the pastpointing opposite to the direction of the vertigo. These spontaneous vestibular symptoms subside within a month after destruction of the end-organ so that in the chronic stage spontaneous vestibular symptoms are absent.

Spontaneous nystagmus, vertigo and reaction movements may be severe in longitudinal fractures and even when a fracture cannot be demonstrated. The intensity of the vestibular symptoms is not a reliable guide for predicting the extent of the damage to the vestibular end-organ.[63] The degree of recovery varies a great deal and progressive deterioration may continue for many months. The vertigo varies in severity, and the vestibular responses likewise may be impaired to a varying degree.[61] In patients with a dead labyrinth there is considerable disability even though the other labyrinth does in time compensate. Patients with such a disability are subject to errors in the sense of location in space when in the dark. Sudden changes in position may bring on an attack of vertigo.

We have attempted to analyze the equilibratory disturbances in the following group of head trauma cases:

1. Longitudinal fracture of the temporal bone.

2. Transverse fracture of the temporal bone.

3. Skull fracture without temporal bone fracture.

4. Head trauma with unconsciousness but without evidence of fracture.

5. Head trauma without unconsciousness or fracture.

Longitudinal Fracture of the Temporal Bone—The caloric test was performed in 17 of 28 cases of longitudinal fracture. A normal response was obtained in nine cases. A complete loss of vestibular function was observed in four cases: two of these had combined longitudinal and transverse fractures, a third had a permanent high tone deafness, and the fourth had a temporary high tone loss which eventually recovered. A hypoactive caloric response was observed on the side of the longitudinal fracture in four cases.

Spontaneous nystagmus, generally mild, was observed in seven of the 28 cases. This sign was observed as late as 10 months after the injury. Dizziness was complained of in 12 cases.

Positional nystagmus was observed in three cases; two with direction fixed, and one with varying direction.

Transverse Fracture of the Temporal Bone — Vestibular reaction to the cold caloric test (ice water) was absent in all 16 cases of transverse fracture. A weak spontaneous nystagmus to the uninvolved ear was elicited after head shaking in seven cases. Eleven of the sixteen cases complained of some disturbances in balance.

Skull Fracture not Involving the Temporal Bone—Out of 13 such cases a normal caloric test was observed in seven; two cases presented a hypoactive caloric response; five complained of dizziness; three had a spontaneous nystagmus; and two cases had positional nystagmus, one direction fixed and the other with varying direction.

Head Injury With Unconsciousness—Fourteen cases were seen in this group. A normal caloric test was obtained in nine cases. An asymmetrical (hypoactive) response was obtained in four cases. Dizziness was present in six of the cases; one was severe. A faint spontaneous nystagmus was observed in three cases. Objective, positional nystagmus was observed in three cases; two were direction fixed and one varying direction. Three additional cases presented a history of bouts of dizziness with change in position, but the positional nystagmus could not be elicited.

Head Injury Without Unconsciousness or Fracture—A total of 32 cases were seen. The caloric test was normal in 16 cases and asymmetrical in five cases. Dizziness was a symptom in 16 cases, four of which were severe. Dizziness was absent in 13 cases. A spontaneous nystagmus was observed in three cases. Positional nystagmus of fixed direction type was observed in two cases.

SUMMARY OF VESTIBULAR FINDINGS.

One hundred cases of head injury were reviewed. Fifty-seven of these had a skull fracture; 50 complained of dizziness; 76 cases had a caloric test. The caloric response was abnormal in 35 cases and normal in 41 cases. Positional nystagmus was observed in 10 cases out of approximately 40 tested.

The abnormal caloric tests consisted for the most part of hypoactive or absent responses on the involved side. Hyperactive responses were not observed. Positional nystagmus was "fixed" in seven of the 10 cases, where it was observed indicating a peripheral vestibular disturbance. In three cases it was of the variable direction type, suggesting a central disturbance. Neurological examination in these three cases failed to show other evidence of a central lesion.

Most of our cases were seen on only one occasion for examination, some many months after the head injury. Consequently, we were not in a position to have a complete clinical picture in each case. Our review showed only 10 per cent of the cases to have objective positional nystagmus. We believe the incidence is much higher.

Usually postural vertigo disappears after a week, or a few weeks. The same holds true for other findings in our review. The material in this study consists of 100 patients who complained of certain or all the symptoms of the vestibulocochlear mechanism. The discrepancies seen in the figures are due to the fact that all did not complain of all possible symptoms. Thus, the 32 patients without unconsciousness or fracture complained of dizziness in 50 per cent; of deafness in 75 per cent; and tinnitus in 43 per cent. It should be pointed out that these patients represent a convalescing group; thus they do not give a total cross-section picture.

ROENTGEN FINDINGS.

The visualization of basal fractures involving the petrous bone by Roentgen studies is often difficult. The presence of a radiating fracture line extending toward the top of the petrous bone may be considered as evidence of a longitudinal fracture of the petrosa. This is particularly true when there is bleeding from the ear. Special views such as Stenver's, Mayer's, Schuller's and Chamberlin's may be of value in demonstrating a temporal bone fracture. The recently developed "magnification" techniques[66] are helpful. In general, few longitudinal fractures involving the temporal bone can be shown in conventional Roentgen studies. The fresher the fracture the more likely will be its detection.

Transverse fractures are more readily visualized. Stenver's view is the projection of choice. This fracture can often be detected many years later because of the failure of the otic capsule to heal, once it is fractured. In this series of 44 temporal bone fractures, 26 had positive Roentgen findings of a fracture. Eight of 16 transverse fractures and 18 of 28 longitudinal fractures were positive; the latter group were vault fractures extending into the base. Roentgen findings were positive in 89 per cent of Gurdjian's and Webster's 1285 cases of skull fractures.[36]

TREATMENT.

Treatment in this class of patient includes the management of the bleeding ear, pneumocephalus, possible meningitis and mastoiditis.

The bleeding ear is best treated by the application of fluffy gauze to the external ear and a bandage to hold the gauze in place. Usually bleeding or cerebrospinal fluid otorrhea stops spontaneously in the course of one to five days. Patients with aural bleeding should be on prophylactic doses of sulfonamides and antibiotics to prevent infection, particularly meningitis.

Meningitis is a serious complication of temporal bone fracture, and was common before the days of sulfonamides and penicillin therapy. The fact that, in many, fractures remain patent for many years following injury, and the fact that these fractures may communicate with preformed pathways giving access to the intracranial spaces, threaten the development of intracranial infections many weeks or months after injury. This has definitely changed since the use of antibacterial therapy. Such patients who develop a meningitic attack have been successfully treated and cured, and this is the rule rather than the exception in this class of patient.

Mastoiditis and otitis media were also common complications following head injury before the days of antibiotic and sulfa therapy. These, too, have been greatly diminished. The presence of such complications should be noted and appropriate treatment given. Persistent mastoid suppuration in spite of antibacterial therapy would call for eradication of the infected bone. Suppuration in the air cells of the petrosa may require surgical exenteration of the involved cells. Prophylactic middle ear exposure is discussed by some otologists but this concept is not fully accepted.[65]

THE FACIAL NERVE.

Incidence of Facial Nerve Injury—In closed head injuries Grove reported 29 cases of facial paralysis among 211 patients with skull fractures involving the ear. In our study of 57 fractures nine had facial paralysis. Gurdjian reported 14 cases of facial paralyses in 129 cases with bleeding from one or both ears, an incidence of about 11 per cent. Two of the patients in this group had bilateral facial paralysis, one survived and one died. The survivor has had complete recovery of function.

The paralysis was complete and permanent in one transverse fracture of the petrous bone and in five with fractures of a longitudinal type, according to the report of Grove. In the remaining 23, the paralyses improved or cleared up completely. Grove stated that all authors agree that facial paralysis occurs more frequently in transverse than in longitudinal fractures. The incidence in the former is approximately 50 per cent. In longitudinal petrous fractures the incidence varies from 10 to 20 per cent. In our series of 57 fractures, facial paralysis occurred once in 16 transverse fractures (recovered) and seven times in 28 longitudinal fractures (two permanent). In our own experience almost all of the patients with facial paralysis improve or clear up completely. The patients with permanent facial paralysis following craniocerebral injuries are rare. We have had only two such examples, one of which was treated by spinal facial anastomosis.

Facial nerve paralysis in lacerations of the face are seen in some cases of craniocerebral injury. We have operated on seven such cases. Four were in lacerations involving branches of the pes anserinus, and three were of lacerated facial trunks proximal to the pes or near the stylomastoid foramen.

Signs and Symptoms—A complete facial nerve paralysis involves an inability to contract the facial muscles of expression on the affected side; the patient is unable to whistle, or to wrinkle the forehead on the affected side. When he is asked to close both lids, the eyeball deviates up and out. The corneal reflex is lost on the involved side, which becomes of practical importance in testing lid reflexes in the comatose patient. If the paralyzed side only is examined, the incorrect conclusion may be reached that the coma is profound. Atrophy occurs as a late result, with a reaction of degeneration present in the muscles of the face and with degeneration fibrillations recordable by electromyography. The progress toward recovery can be estimated by these methods. Rarely a bilateral complete facial paralysis occurs.

Facial paralysis of the peripheral type may occur immediately at the time of impact or it may be seen four or five days after injury. The type that begins several days after injury usually is recovered from quite satisfactorily.

It is important to identify a peripheral facial paralysis from a central type of facial paralysis. In the central type there is usually activity of the forehead and the eye, with serious involvement of the musculature about the mouth. Electrical reactions are also normal, but this is of little aid since the reaction of degeneration does not occur until about 14 days after the onset of the paralysis. This type of facial paralysis is, of course, on the contralateral side from the cortex or pyramidal system involved.

The facial nucleus in the pons may be damaged in head injury resulting in a unilateral peripheral type of paralysis. Other cranial nerve nuclei or central tracts may also be involved in such deep seated lesions, characterized by multiple intrapontile hemorrhages. Single massive hemorrhages also have been observed in patients who may have associated lesions, such as a subdural hematoma with uncal herniation resulting in anoxia with hemorrhages from increased permeability of the vessels.

Occasionally a masking of the face may be seen in the chronic stage of head injury due to a mimetic or emotional facial weakness. This may result from deep-seated damage, probably involving extrapyramidal connections with the facial nucleus, as may occur in Parkinsonism. The patients manifest diffuse brain injury associated with pyramidal tract signs, and may have survived a long unconscious state.

Pathology—The paralysis as stated previously may be immediate or may appear as late as several days to two weeks after injury. An immediate onset usually indicates physiological or anatomical nerve injury in the Fallopian canal, or at the internal auditory meatus. A delayed onset of the facial paralysis usually is due to an edema of the nerve associated with the reparative processes at the fracture site. Small hemorrhages within the nerve sheath with edema may also be a cause for this condition. In many instances nerve function returns, and this has been attributed to recovery of the nerve from injury by stretching, contusion, interfascicular hemorrhage and even section. It appears possible that a torn facial nerve under favorable conditions may regenerate. Ischemia of the nerve from edema has also been suggested as a cause for the paralysis. Cases of bilateral facial paralysis are rare but have been seen, and might be more frequent, if some of the more seriously injured individuals survived long enough for careful neurological examination.

Recovery is almost always the end-result in patients with facial paralyses due to closed head injuries of the head. At times there may be partial recovery with marked stiffening of the face on the affected side, and mass movements of the entire face such as in laughing or in expressive activities. This may be associated with evidences of twitchings and fibrillations of muscles in the facial group. If recovery is complete, it usually follows a short duration of facial paralysis. Facial paralysis which persists longer than three months usually will improve in part only. If the aforementioned, fibrillations and mass movements of the face on emotional activity are present and if recovery does not occur within 18 months to two years, one should consider the advisability of spinal facial anastomosis or hypoglossal facial anastomosis. We are not impressed that in the greatest majority of cases decompression of the nerve in the facial canal would be of value, since a majority of these patients recover completely, or almost completely, in a reasonable period of time, and results of improvement after operation should not necessarily be construed as evidence that the operation is effective.

Open Injuries—In craniocerebral injuries facial nerve paralysis due to laceration of the nerve on the face or near the mastoid area poses a definite problem.

The nerve or its branches may be involved anywhere on the face. Shell fragment wounds near the ear and the mastoid area may destroy a segment of the nerve, presenting little hope for reconstructive surgical treatment of the nerve. Damage following mastoid surgery may produce anatomical destruction of the nerve trunk. Knife wounds, lacerations on the side of the face and injury associated with the removal of parotid tumors may cause extensive injury. Lacerations near the mastoid area may produce a simple division of the trunk, and in some instances there may be an involvement of the branches of the pes anserinus as well as the parotid duct.

Treatment—In closed head injury, treatment of facial paralysis of the peripheral type is always conservative, employing physiotherapy with massage, mild heat and exercise of the facial muscles. The prospect for recovery is promising in the greatest majority of cases. By means of the electrical responses of the facial muscles to faradic and galvanic currents, observations may be made which will aid in prognosticating the outcome or the need for further therapy. A period of about nine months should be allowed for spontaneous recovery. Operative intervention in our hands is not employed until 18 months have elapsed without any improvement in the facial function.

Open injuries, the result of shell fragments or bullet wounds, may be of such massive degree as to preclude repair of the facial nerve in any part of its course. Lathrop has found it possible to deal with this type of injury in some cases with considerable success by the use of nerve grafts.

Stab wounds and lacerations of
offer the possibility of complete r
the nerve can be anastomosed. A m
in this type of injury is exploratio
immediately after injury. This is
branches of the facial nerve periph
When the branches have been sect
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the wound for exploration. [illegible]
ments.

When irreparable injury has involved the central portions of the nerve, the use of a facial hypoglossal or facial spinal accessory anastomosis is useful. A degree of tone develops in the facial muscles which encourages symmetry in the resting face. Voluntary movements of the face are dependent upon associated shoulder or tongue movements. Some patients become very agile in the use of the associated movements to bring about expressive changes of the face.

Nerve grafts may be considered in the treatment of cases not suitable for direct nerve anastomosis. Plastic procedures employing fascial slings have been found helpful, or a buccal fascial splint which is attached to the upper molar region on the paralyzed side may be useful.

SUMMARY AND CONCLUSIONS.

1. The types of blows commonly associated with fracture of the temporal bone are reviewed and classified. Temporoparietal area blows spread the skull in an antero-posterior direction producing a vertical fracture in the lateral aspect of the skull which may extend toward the base of the skull, producing a longitudinal fracture of the temporal bone.

2. Occipital area blows spread the skull from side to side fracturing it vertically near the midline. The fracture may extend forward to the middle fossa and fracture the petrosa transversely, producing a loss of cochlear and vestibular function and frequently damage to the facial nerve.

3. Frontal area blows also spread the skull from side to side fracturing it vertically near the midline. The fracture may extend posteriorly to the base of the skull in the anterior, middle, and even posterior fossae, resulting in a transverse fracture of the petrosa.

4. The types of blows producing isolated fractures of the base are reviewed.

5. The pathologic findings in longitudinal and transverse fractures are summarized.

6. Early experimental and pathological studies of temporal bone fractures emphasized hemorrhage into the labyrinth as the primary cause of cochlear and vestibular disturbances. Recent investigations suggest that neural degeneration in the membranous labyrinth is due to shock or a pressure pulse set up in the inner ear fluids by the head blow. Intense stimulation produces a violent displacement of the basilar membrane, causing injury to the organ of Corti. The same stimulus may disrupt the otolithic membrane on the end-organs of the utricle and saccule resulting in disturbances referable to those sense organs. Hemorrhage into the labyrinth in the light of recent studies does not appear to be responsible *per se* for deafness or for vertigo.

Hearing losses following head trauma are rarely if ever attributable to central lesions. Bilateral destruction of the auditory cortex does not create a threshold loss for hearing. Up to 75 per cent of nerve fibers to a particular region of the cochlea can be destroyed without creating threshold hearing losses for frequencies localized in that region, and the constant presence of loudness recruitment supports the concept that head trauma damages the organ of Corti.

7. Repeated audiograms are necessary to properly assess the end result of head trauma on hearing.

8. Dizziness following head trauma may be either peripheral or central in origin. In transverse fractures there may be a severe vertigo resulting from destruction of the membranous labyrinth. Postural vertigo is believed to result from injury to the utricular macula and its associated otolithic membrane. The central type of dizziness is a sensation of disequilibration, and not one of true rotation. The term "blacking out" is the most descriptive. It is suggestive of brain stem involvement (vestibular nuclei and fibers, tecto-spinal, rubro-spinal and spino-cerebellar pathways).

9. In closed head wounds pressure gradients build up at the craniospinal junction resulting in shear and tear of the elements in the brain stem. In open head wounds the forces of injury are quickly dissipated, the brain stem is usually not injured, and dizziness does not usually occur.

10. The symptoms and findings in central involvement of the vestibular mechanism are reviewed.

11. Postural vertigo and positional nystagmus are frequent findings in cases of head trauma. Injury to the peripheral vestibular mechanism results in a positional nystagmus of fixed direction. A direction-changing nystagmus suggests a disturbance in the central vestibular nuclei and pathways.

12. Cupulometry has recently been reported to offer quantitative data on the function of the vestibule with respect to nystagmus and the sensation of vertigo.

13. The findings on clinical examination of head trauma cases are often temporary. Many times these cases are seen for only one examination. It is only with repeated and thorough examination that a complete clinical picture is obtained.

14. A thorough and painstaking Roentgen examination is required to demonstrate temporal bone fractures. The recently developed magnification techniques are recommended where available.

15. Treatment of temporal bone fractures is conservative. A prophylactic use of antibiotics and sulfonamides is advisable. Aural bleeding can often be controlled by merely having the patient assume a sitting position. Threatening suppurative complications may call for appropriate surgical intervention.

16. Most cases of facial paralysis clear up spontaneously. Surgical intervention is seldom necessary. Treatment is conservative with physiotherapy, massage and exercises.

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SIALOGRAPHY IN THE DIAGNOSIS AND TREATMENT OF LESIONS OF THE PAROTID GLAND AND DUCT.

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Since the publication of a paper on sialography[1] by one of us ten years ago, the employment of this procedure has frequently facilitated the diagnosis and treatment of lesions of the salivary apparatus. The technique as there described has required no essential modification; however, valuable experience has been gained in the interpretation of Roentgen shadows, especially those characteristic of uric acid calculi.

Parotid injection is a comparatively simple procedure and apparently devoid of danger. We have on occasion seen the duct perforated and lipiodol injected into the surrounding tissues without harmful results.

Injection is best performed by reflected light with the patient reclining upon the X-ray table. A 5 cc. glass syringe fitted with a blunt 3/8 inch 20 gauge needle simplifies the procedure. In order to decrease the viscosity of lipiodol, the "loaded" syringe with needle attached is allowed to remain in a hot water bath until the time of injection. Steno's duct, which makes an almost right angle turn as it passes over the masseter muscle, is now straightened by pulling the cheek outward and forward. The mouth of the duct is then dilated by means of No. 1 and No. 2 lachrymal probes, followed at once by the slow injection of 1. - 1.5 cc. of warm lipiodol. A small wad of cotton is placed over the papilla so as to prevent leakage and confusing shadows. Roentgenograms are taken in lateral, oblique, and P. A. positions.

Interpretation of sialograms may require some training, but with a little practice characteristic shadows may be ob-

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served in uric acid calculi, fistulae, strictures, sialectasis, mixed tumors, malignancies and extra-glandular lesions; but, the most practical value of sialography lies in differentiating mixed and malignant tumors, in the accurate determination that malignant degeneration of a mixed tumor has taken place.

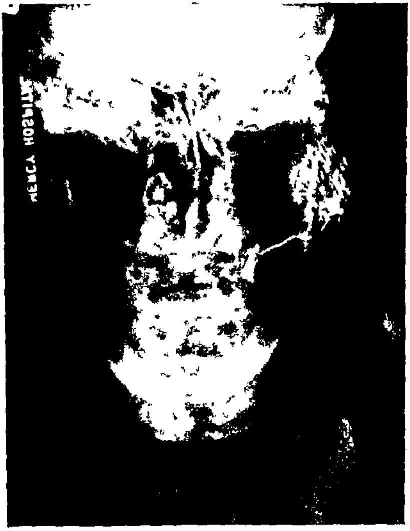

Fig 1 Normal sialogram Note uniform caliber of duct

Note in the sialogram of a normal parotid, the uniform caliber of the duct (see Fig. 1.) and the arborescent character of the gland parenchyma (see Fig. 2). In the oblique position an accessory gland is clearly visible (see Fig. 3). In mixed tumors the duct is apt to be displaced forward and downward (see Fig. 4) due to what the radiologist calls a space-occupying mass. The substance of the gland, however, fills perfectly.

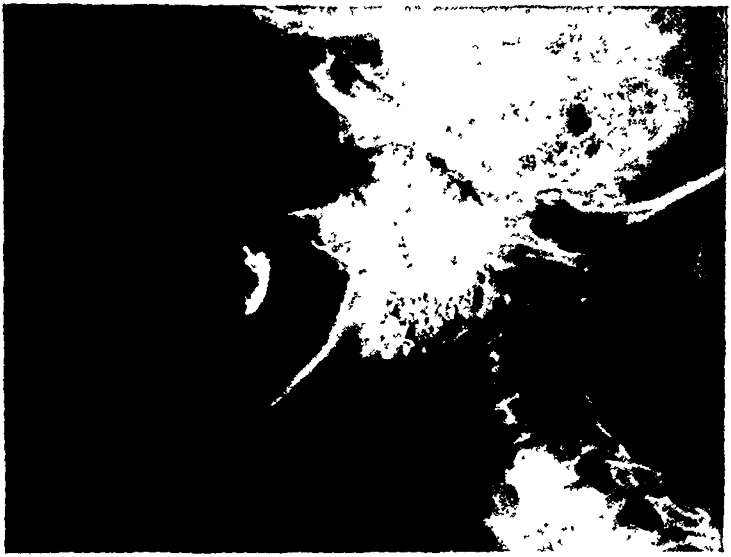

Fig. 2. Same as Fig. 1. Note arborization of gland parenchyma

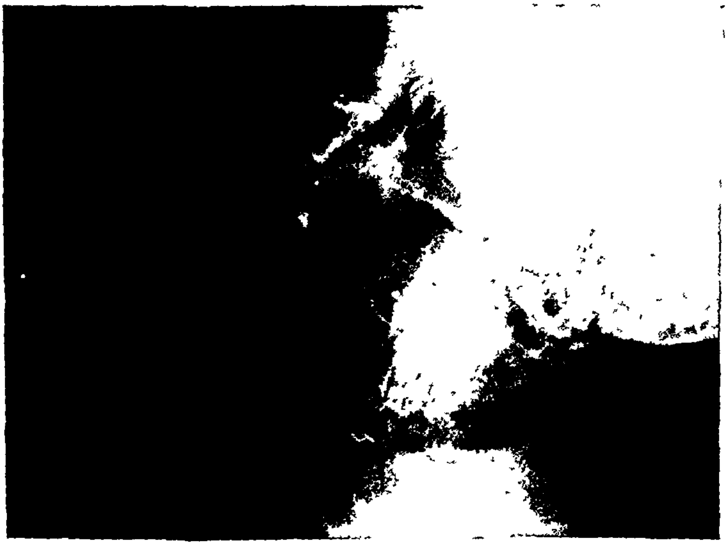

Fig. 3. Same as Fig. 1. Accessory parotid gland above Steno's duct.

In malignancies the glandular parenchyma is destroyed completely (see Fig. 5). In such cases the accessory gland may greatly hypertrophy and lead to error in diagnosis.

Besides facilitating the differential diagnosis of salivary lesions the injection of iodized oils has at times proven to be of definite therapeutic value. Idiopathic swellings of the parotid not infrequently subside promptly. Whether this is due to bactericidal action, the increased flow of saliva or lubrication has not been determined, but the fact remains that following this simple procedure parotid swellings often vanish readily, and permanently.

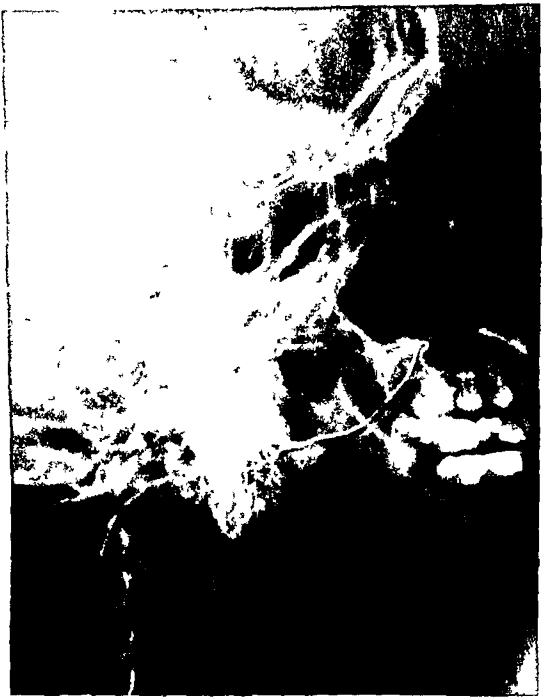

Fig 4 Mixed tumor Note (a) Hairpin curve of Steno's duct, due to 'space-occupying mass' pushing duct forward (b) Accessory parotid and duct just above Steno's duct (c) Normal filling of gland parenchyma

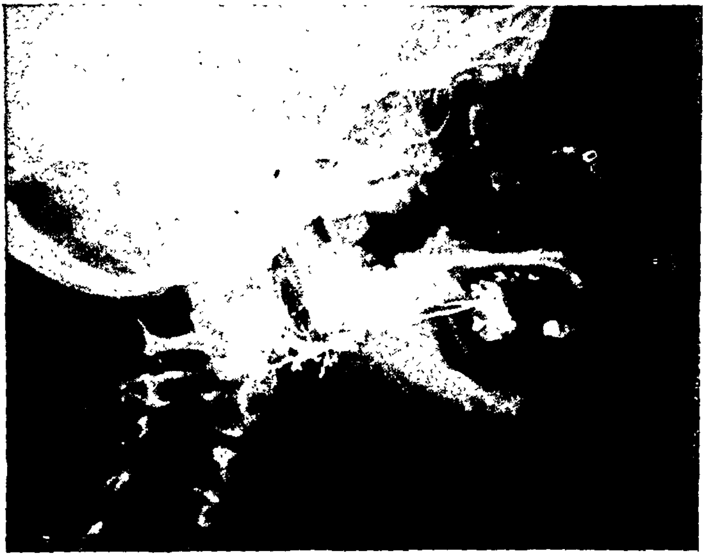

Fig. 5. Sialogram of Fig. 12. Malignant mixed tumor. Note parenchyma of main gland destroyed. Accessory gland greatly hypertrophied.

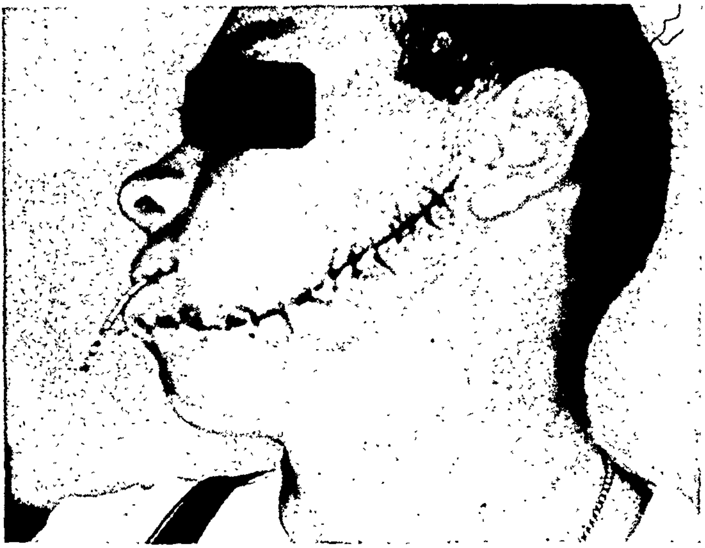

Fig. 6. Salivary fistula due to razor cut. Repair followed by retention catheter. Improved method of anchoring catheter described in text.

The treatment of lesions in and about the parotid has been materially advanced by sialography and the use of retention catheters. Aside from infectious parotitis the most common lesions encountered are calculi, mixed tumors, fistulae, strictures and malignancies in the order given. The surgical treatment of calculi[2] and malignancies of the duct[3] have been described elsewhere.

Masseteric parotid duct fistulae (see Fig. 6) may be readily closed by isolating and carefully coapting the cut edges of the divided duct with fine catgut and inserting a retention catheter (see Fig. 7) which is allowed to remain *in situ* for two to three weeks. Re-establishing the normal channel in this manner has distinct advantages over older, more uncertain, methods of converting external into internal fistulae.

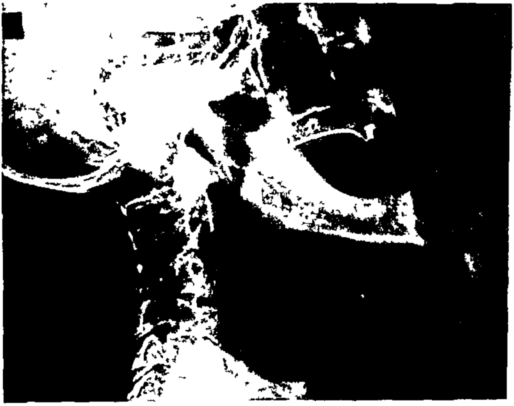

Fig 7. Catheter in Steno's duct fixed to cheek by means of skin clip

Strictures of the duct (see Fig. 8) which do not respond to dilatation may be dealt with in a similar manner. The constricted portion of the duct is resected, and the freshened proximal and distal ends are united by fine catgut sutures, followed by retention catheter.

responsible. The common tendency of radiating all salivary tumors should, therefore, be strongly deprecated.

Mixed tumors grow slowly. They are always encapsulated and may attain great size (see Figs 9 and 10). Because of the danger of malignant change all mixed tumors should be carefully extirpated. In small or moderately sized tumors, an incision made over the most prominent part, in the line of the facial nerve fibers or Steno's duct, will rarely injure

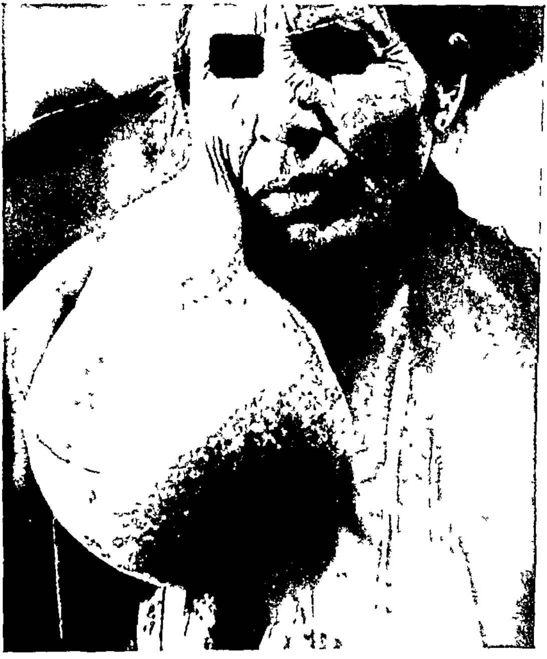

Fig 9 Mixed tumor of the parotid Pueblo Indian showing size mixed tumor may attain without malignant degeneration

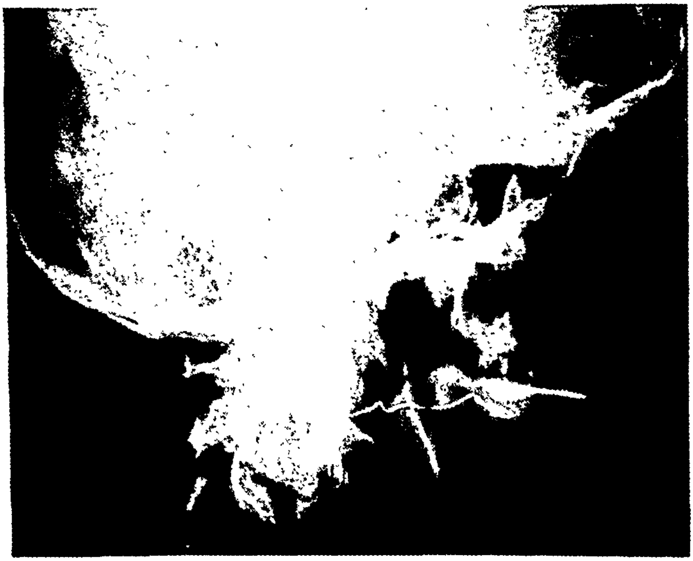

Fig. 10. Calcifying muco-epidermoid adenoma of right parotid.

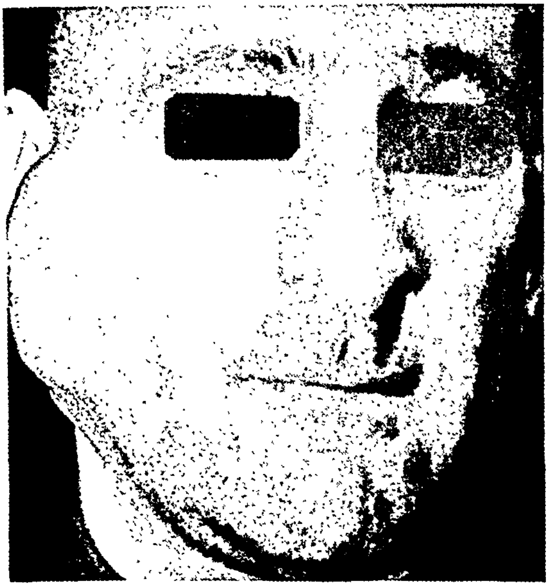

Fig. 11. Malignant mixed tumor. Squamous-cell carcinoma Grade 4. Radical resection, 1941. Patient survived 8 years in spite of refusal of neck dissection.

these structures, nor is there much danger of salivary fistula in carrying the incision right through the substance of the gland To safeguard against this eventuality a catheter may be left *in situ* The accessory parotid must, of course, be identified and avoided

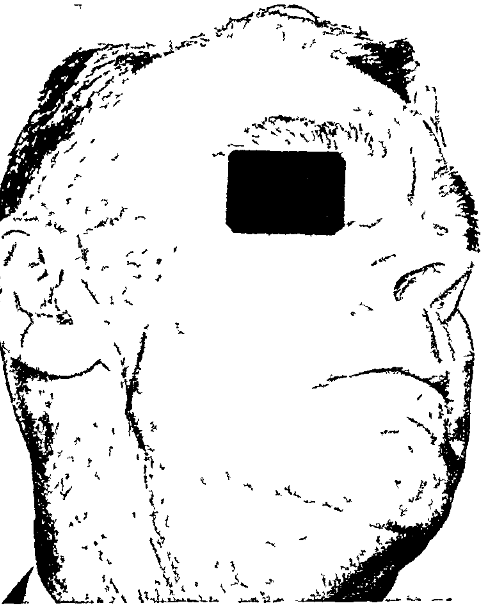

Fig 12 Parotid duct calculus mixed tumor and malignancy in the same patient Radical resection in 1949 Scars in cheek due to fascial implants (elsewhere) which became infected Patient well since December 1949

Before leaving the subject of mixed tumors, attention should be drawn to an article published in the *Archives of Otolaryngology* (Aug 1953) Mumma[4] reports the case of a mixed tumor of the tongue successfully treated by the injection of colloidal chromic phosphate (CrP_3O_4) "There has been no *recurrence in three years.*" *This is an encouraging report that* augurs well for the treatment of these lesions by radio-active chromic phosphate.

Malignant tumors of the parotid (see Figs. 11 and 12), whether primary or secondary, generally demand radical resection with sacrifice of the facial nerve. Prolonged efforts calculated to preserve the integrity of this structure appear to us as non-surgical. Radical parotid surgery may be a life-saving measure. The resulting facial paralysis is clearly outweighed by the patient's chances of recovery; besides, fascial implants are now successfully employed with excellent cosmetic results. Without going into the details of technique it may be noted that ligation of the external carotid greatly facilitates the operation.

In the employment of contrast visualization techniques preliminary to radical surgery of the parotid a word of caution may not be amiss. The value of sialography as an operative determinant must not be overrated. Deliberate sacrifice of the facial nerve is probably never justified by Roentgen findings alone; fortunately, in most instances, other signs and symptoms are present which corroborate the diagnosis. Sudden rapid growth and pain in a dormant mixed tumor are highly significant. Infiltration, local fixation of the skin and cervical adenopathy are ominous signs which suggest the true nature of the lesions. Façial paralysis attests that the nerve has become infiltrated by the malignant process.

CONCLUSIONS.

Sialography has become a valuable adjunct in the diagnosis of lesions of the salivary apparatus.

In idiopathic swellings of the parotid the injection of iodized oil may be of great therapeutic value.

The use of the retention catheter is of value in the repair of salivary fistulae and strictures as well as in the prevention of ductal strictures.

The greatest value of sialography lies in differential diagnosis of mixed and malignant tumors.

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227 16th Street

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Arrangements have been completed for the joint meeting of the North Carolina Society of Eye, Ear, Nose, and Throat, and the South Carolina Society of Ophthalmology and Otolaryngology September 17, 18, 19, 1956. Headquarters will be the George Vanderbilt Hotel, Asheville, North Carolina

An unusually attractive program has been arranged, and a large attendance is anticipated.

Asheville, North Carolina, is in the mountains of Western North Carolina, and is a particularly beautiful spot in this season of the year.

For further information write Roderick Macdonald, M. D., Sec. and Treas., 330 East Main Street, Rock Hill, S. C.

STRICTURE OF THE EXTERNAL AUDITORY CANAL.

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The purposes of this paper are to present the information gained from the study of eight cases of acquired closure of the external auditory canal and to describe a simple endaural technique for the correction of this deformity. Numerous papers[1,2,3,4,5,6] concerned with agenesis, or aplasia of the ear canal, have appeared in the otolaryngologic literature, but few recent publications have dealt primarily with closure of acquired origin.

On occasions, the nomenclature of the two entities has been confusing. House[7] agreed with Kinney[8], who referred to congenital absence of the meatus as "aplasia", and felt that the term "atresia" should be reserved for acquired stenosis. "Atresia", however, is a Latin word; but it is derived from two Greek words, "A" and "tretos". The latter means "to perforate". The literal translation, then, of "atresia" is "against perforation" or "without perforation," and would not necessarily indicate a canal which had been once open and subsequently closed. Perhaps the word "stricture" from the Latin "stringere"—to draw tight, is more apropos for the abnormality under consideration.

Cohen and Fox[9] believed that ear canal stricture was rare, having observed it in only two out of 17,000 cases, in which care of the ear, nose and throat was sought. Ballenger and Ballenger[10] suggested ear canal cartilage atrophy, with subsequent wall collapse, as an occasional cause. Novick[11] reported a case which was probably due to the careless use of caustics or curettes. Two cases of stenosis, accompanied by mastoiditis and cholesteatoma, were reported by Lillie and McBean;[12] and they advocated surgery to prevent intracranial complications in such cases. The use of split thickness skin grafts with sponge packing, to ensure patency, was suggested by Conley[13].

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* From the Department of Otorhinolaryngology of the University of Kansas School of Medicine.

ANALYSIS OF CASES.

The Chief Complaint.

Eight cases of true meatal stricture were encountered in our outpatient department in the past three years. Two patients complained of postauricular fistulae, one of headache on the involved side, one of vertigo, three of pain in the ear, and one of cosmetic deformity. None have hearing loss as the chief complaint, although all admitted to it on questioning. The duration of difficulty varied from six months to 19 years. One had been previously operated without success.

The Cause.

Three of the strictures were produced by burns—one chemical and two thermal. One was the result of a football injury, and one followed a razor wound. Two were due to surgical procedures performed for the removal of parotid cysts or tumors, and one was incident to an automobile accident.

Radiologic Findings.

In seven cases mastoid destruction to some degree was demonstrable by X-ray, while the uninvolved side invariably exhibited normal pneumatization. An area of rarefaction in the region of the antrum, suggesting cholesteatoma formation, was a surprisingly common feature (five cases).

Hearing Loss:

Since most of the patients were suffering from infection and bone destruction, it was felt that the hearing was of secondary importance; nevertheless, air and bone conduction determinations were done on all cases. As one might expect, those of several years' duration possessed hearing curves typical of mixed type deafness. Those of more recent origin showed a conductive type loss, but the hearing in every involved ear was below the 30 db level for all tones in the conversational range. Two older individuals had high tone loss on the uninvolved side.

Physical Findings.

With one exception there was complete fibrous closure of the canal in the outermost portion. In one instance it was

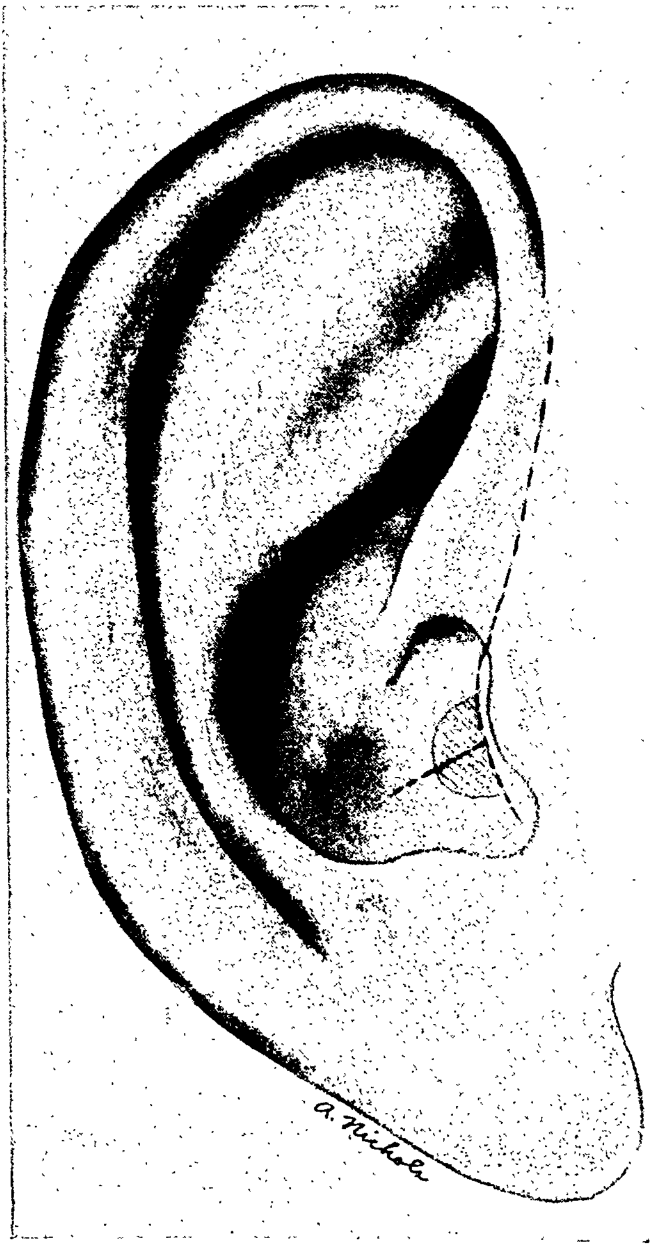

Fig. 1. The Incision.

possible to pass a small probe into the lumen for at least one cm. A probe could also be introduced into the postauricular fistulae previously mentioned, and cortical dehiscences were thus palpated.

The patient, who had sustained the football injury, had also suffered loss of the lobule. Retroauricular tenderness was present in two of the cases, and in one of these, nystagmus of the second degree was demonstrable. The neck of one individual was badly scarred as a result of the same burn which was the genesis of the stricture. Otherwise, all were in reasonably good health.

Treatment.

All eight cases were treated surgically by a technique which embodied certain of the principles used by both Cohen and Fox, and Conley, yet it differed in some repects from the operative pattern followed by any of these authors. The steps of the operation were as follows:

1. A skin incision was made just anterior to the helix and parallel to this structure. It extended from the superior point of attachment of the helix to a position just posterior to the superior margin of the tragus.

2. Hugging the posterior margin of the tragus, the same incision was extended inferiorly to the region where the inferior wall of the ear canal would ordinarily be.

3. From the center of the post-tragal portion of the above incision another incision was made in a posterior direction and at right angle to the first. It terminated at a point where the posterior canal wall would ordinarily be (see Fig. 1).

4. Two triangular flaps of skin and underlying fibrous tissue were thus created, one above and one below; and the subcutaneous tissue was resected from these flaps (see Fig. 2).

5. Any existing mastoid or middle ear disease was then eradicated by the customary endaural method.

6. A No. 000 silk suture was introduced postauricularly opposite the superior triangle of skin, brought out through the base of the triangle, reintroduced into its apex and re-

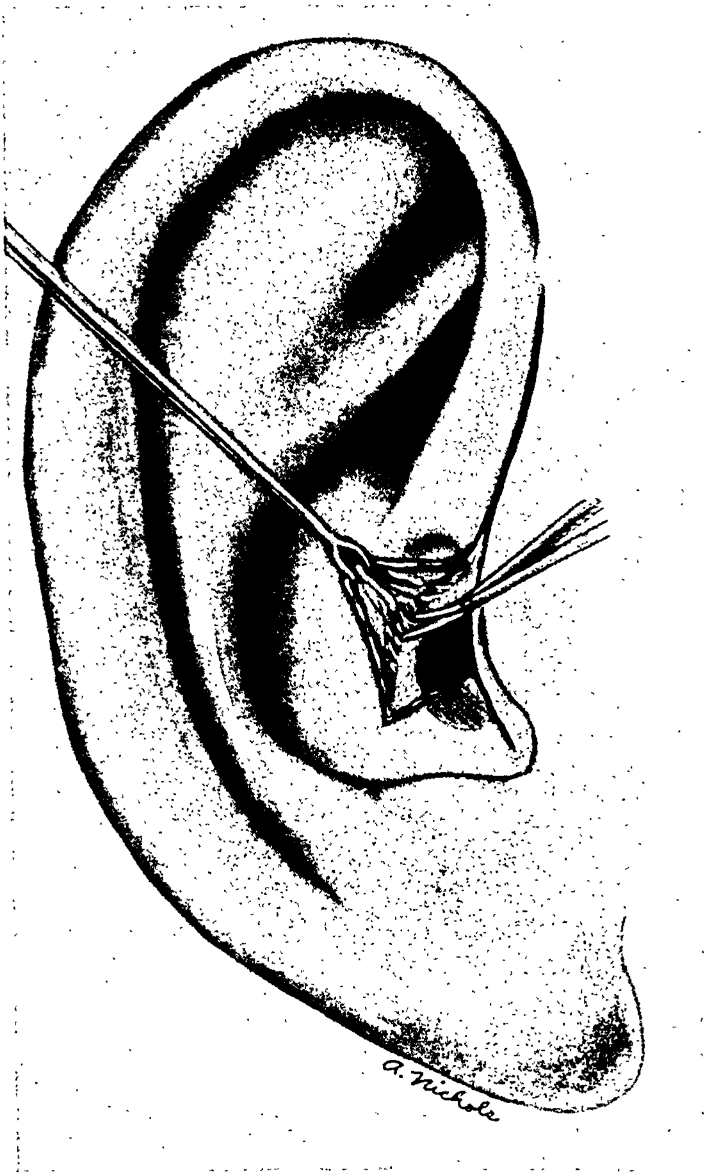

Fig. 2. Removal of Subcutaneous Tissue.

turned to the postauricular area about 0.5 cm. superior to the point where it first entered the skin. The two ends were then tied without tension, thus approximating the superior flap in juxtaposition to the canal. This suture emulated the one used by Sooy[14] to maintain patency of the meatus in the fenestration operation.

7. A second postauricular suture, similar to the first, was used to apply the inferior flap to the posteroinferior canal wall (see Fig. 3).

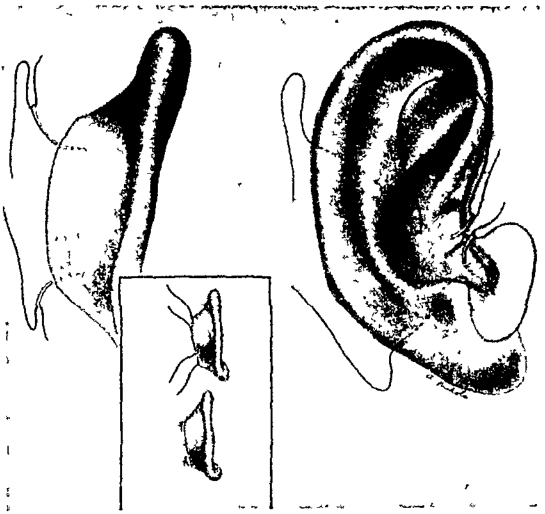

Fig 3 Suturing

8. A split thickness skin graft, obtained from the upper thigh, was applied to the mastoid and middle ear cavities and packed firmly in place with continuous grease gauze.

9. The supratragal portion of the incision was closed with interrupted No. 00000 silk sutures (see Fig. 4).

10. Packs and sutures were removed on the sixth postoperative day.

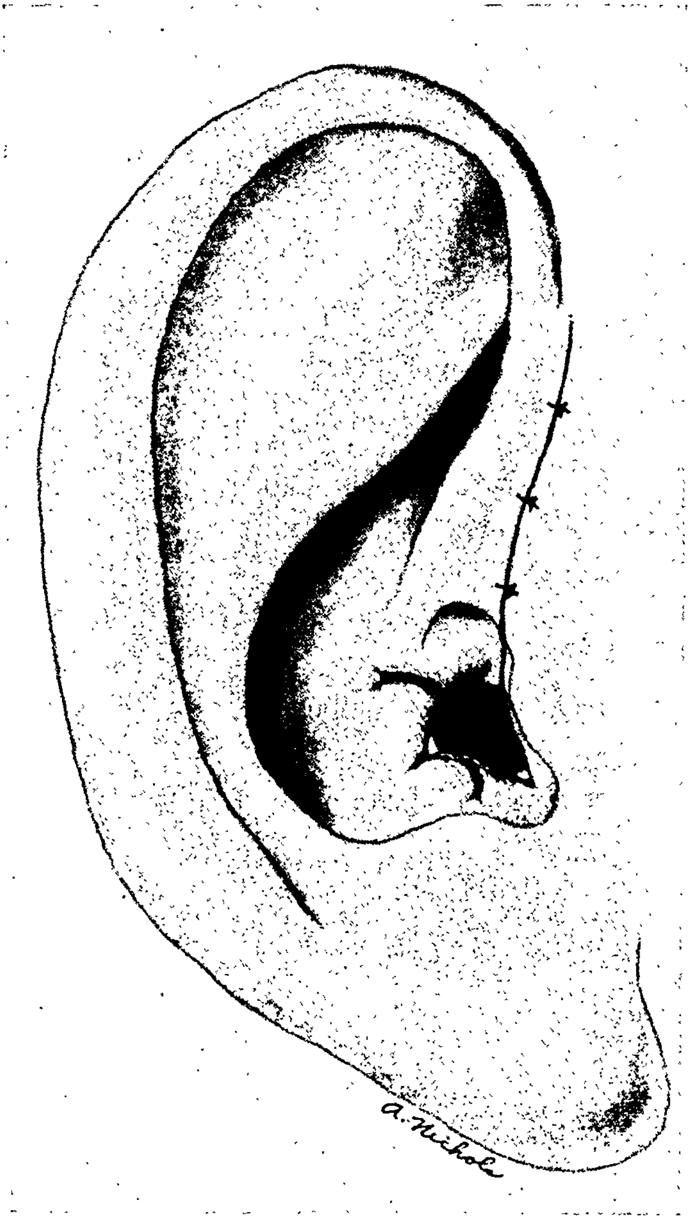

Fig. 4. Incisions Closed.

Operative Findings.

In most cases the extent of mastoid and middle ear disease was surprising, for no case was free of infection. Cholesteatoma was present in three canals, and there were cortical dehiscences in two instances. A fistula was present in one horizontal semicircular canal. Wide dural exposure was evident in one operation, and it amounted to almost complete absence of the tegmen mastoidea.

There was only one example of minimal infection, and that was apparent in the case of only six months' duration. Here, the membrana tympani was still intact, and the mastoid process was not diseased; nevertheless, the canal was full of pus, and it was assumed that more serious trouble lay ahead.

Results.

Permanent patency of the external auditory canal was obtained in all patients, and the cosmetic results were invariably good. Six of the ears became completely dry, but two still form granulation tissue and mucoid discharge. Both of the cases with postauricular fistulae were troublesome.

Although each fistula was carefully closed at the time of surgery, both reopened within two weeks. Secondary closure by marginal skin excision, undermining and suturing was successful. Improvement in hearing was realized by only one patient, for in all others the condition had been present for so long that extensive middle ear disease had developed.

ILLUSTRATIVE CASE.

J. B., a 63-year-old colored male, was first seen in the outpatient department on Dec. 30, 1953. He stated that he had sustained a severe burn of the left side of the face in 1938, and from that time on the ear canal had progressively narrowed. If finally closed entirely in 1948, but on occasions a bead of foul smelling pus would appear. Severe aural pain began two months prior to his outpatient visit.

The past history included mild attacks of asthma of 20 years' duration, and was otherwise irrelevant.

The only significant finding on physical examination was complete fibrous stricture of the left external auditory meatus, associated with moderate scarring of the auricle (see Fig. 5). X-ray revealed destruction in the mastoid process and an area of rarefaction anterior to the lateral sinus (see Fig. 6).

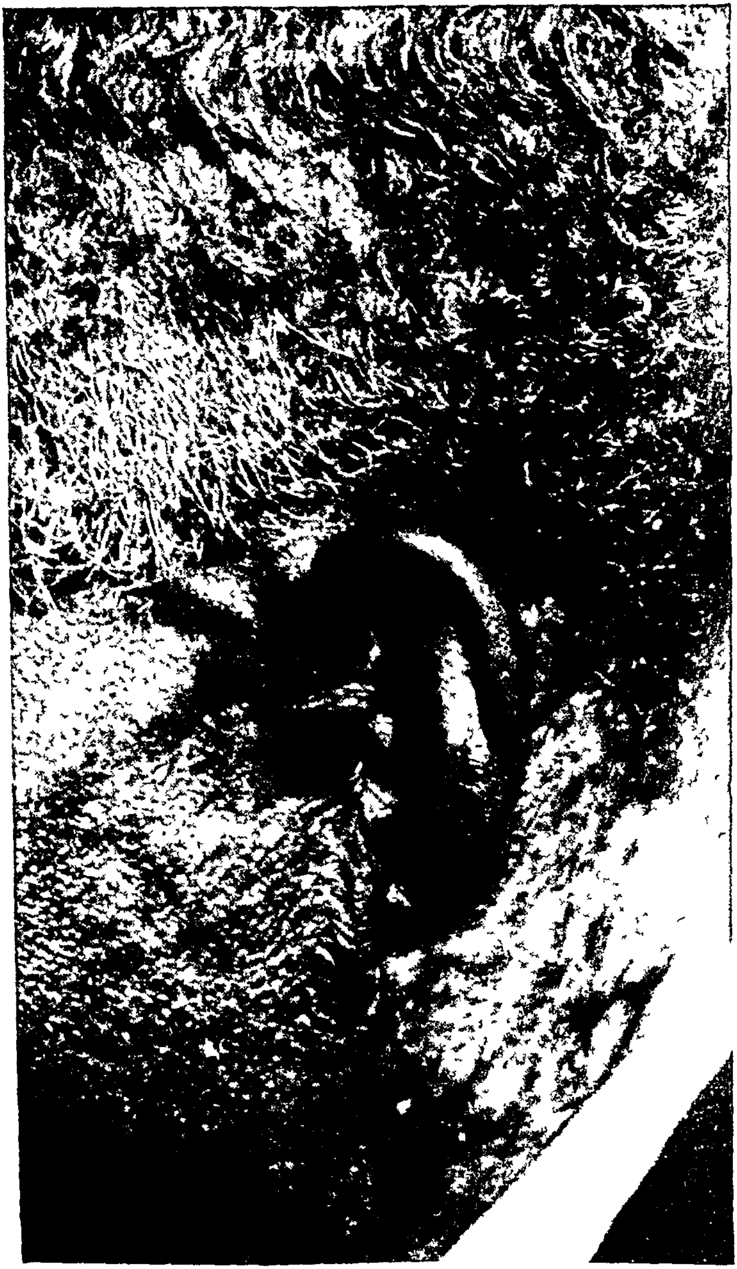

Fig. 5. Preoperative Stricture.

An X ray of the chest revealed a fibrotic lesion of the right apex This was diagnosed as quiescent tuberculosis, since on repeated sputum examinations no tubercle bacilli were found The EKG presented no abnormality, and all laboratory findings were within normal limits

On March 2 1954, he was admitted to the hospital, and surgery was performed two days later The procedure was identical to that described in the context, and pus and necrotic bone were found in the mastoid process A modified radical mastoidectomy was done, and the mucocutaneus flap was used to cover the medial antral wall A split thickness skin graft, prepared from the upper thigh, was applied to the temporalis muscle tegmen mastoidea and facial ridge The skin incision was closed after packing the cavity with aureomycin ointment gauze

Fig 6 Mastoid X ray

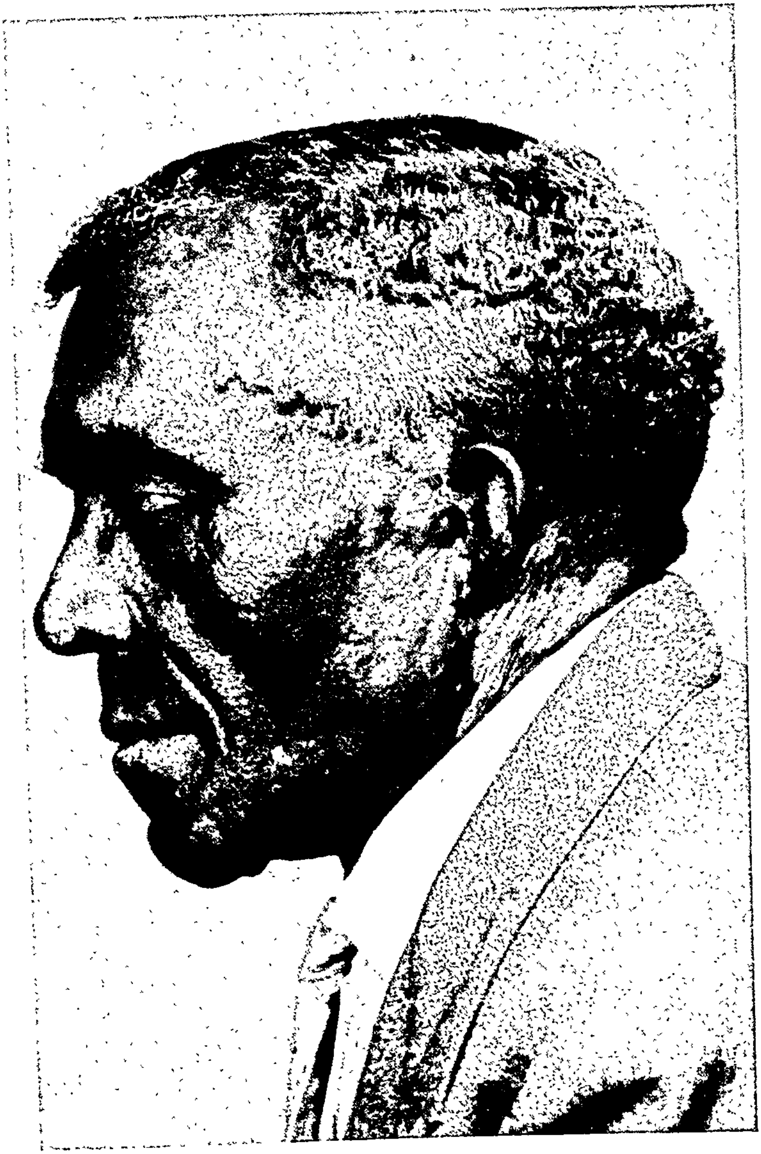

Fig. 7. Postoperative Result.

The postoperative course was uneventful, and the pack and sutures were removed on the seventh postoperative day. All discharge had ceased six weeks later and canal patency has been maintained until the present time (see Fig. 7).

DISCUSSION

Appraisal of the operative findings would seem to indicate that any acquired external auditory canal closure may well be dangerous to the patient The acquired type is probably more apt to be so, because the object which causes the wound will usually introduce infection into the canal or middle ear Unless associated with a primary cholesteatoma, aplasia of the ear canal is less hazardous unless a previous unsuccessful attempt at surgical correction has been undertaken

It would appear that radiographic studies of the temporal bone are of great value, not necessarily to establish the presence or absence of an ear canal, but also to determine the existence and extent of mastoid disease

The endaural repair offers the advantages of simplicity and good cosmetic result, and it provides an epithelial lining of the meatus, furthermore the lines of stress, so created, favor permanent patency

CONCLUSIONS

1 Mastoiditis, middle and inner ear infection and postauricular fistula are complications of stricture of the external auditory canal

2 The deformity deserves early surgical correction

3 An endaural approach, resulting in a complete epithelial canal lining, may be utilized

4 New lines of stress will aid in maintaining canal patency

SUMMARY

An analysis of eight cases of stricture of the external auditory canal is presented, and an endaural approach for the correction of the condition is described

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DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PROGRAM 1956

Tuesday, February 7, 1956

Amon Carter Airport—Private Dining Room

Joint Meeting Fort Worth E.E.N.T. and Dallas O. and O.
Dr. Frank B. Walsh
Baltimore, Maryland

4:00 P.M.—Program:
Tumors of the Optic Nerve and Chiasm

5:30 P.M.—Cocktails, courtesy Alcon Laboratory

6:30 P.M.—Dinner honoring Dr. Walsh

7:30 P.M.—Myasthenia Gravis

THE DIAGNOSIS AND TREATMENT OF UPPER RESPIRATORY SYMPTOMS PRODUCED BY INCREASED VISCOSITY OF MUCUS AND DRYNESS OF THE MUCOUS MEMBRANES[*]

JACK R ANDERSON, M D ,

and

WALLACE RUBIN, M D ,

New Orleans, La

Because otolaryngologic thinking is so oriented in terms of allergy and infection, there is a tendency to overlook the fact that in approximately 20 per cent of the cases, symptoms accompanying upper respiratory disease are caused by increased viscosity of mucus and varying degrees of dryness of the mucous membrane instead of hypersecretion or abnormal fluidity as is generally assumed

The true nature of the condition can be established by taking a good history, performing a thorough physical examination, and conducting a cytologic study of the secretions

The purpose of this presentation is to review the diagnosis of this condition and to report the use of intranasal histamine in its treatment

SYMPTOMATOLOGY

Table I is a compilation of the more frequently encountered complaints due to increased viscosity of the mucus and varying degrees of dryness of the mucous membrane of the upper respiratory tract We have used verbatim quotation to emphasize how truly common these symptoms are in the average otolaryngological practice, and to call attention to the fact that they may strongly mimic those due to diametrically opposed conditions, *viz* , hypersecretion, or abnormal fluidity

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[*] From the Ear Nose and Throat Allergy Clinic of the Eye Ear Nose and Throat Hospital New Orleans Louisiana

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TABLE I.

FREQUENTLY ENCOUNTERED SYMPTOMS DUE TO INCREASED VISCOSITY OF THE MUCUS, AND VARYING DEGREES OF DRYNESS OF THE MUCOUS MEMBRANE OF THE UPPER RESPIRATORY TRACT.

A. NASAL SYMPTOMS:

1. Blockage of the nose.
2. Dryness inside the nose.
3. Burning sensation in the nose.
4. Tickling in the nose.
5. Sinus trouble.
6. My nose is always blocked and nose drops give me relief for only a short time.
7. The only relief I get for my nose blockage is to wash it out every day with salt water.
8. The only way I can get relief of my nasal blockage is to have my nose packed.
9. I don't sneeze often, but when I do and my nose runs a little, I feel much better.
10. A big glob of mucus accumulates in my nose, and I feel terrible until I can work it loose.
11. At times my nose gets dry and starts to bleed a little.

B. OROPHARYNGEAL AND PHARYNGEAL SYMPTOMS.

1. I have a dripping from the back of my nose into my throat.
2. I have to "hawk" a great deal to clear the back of my nose.
3. I think my palate has fallen; or, I feel as though that little thing hanging from my palate is too long and is lying on my tongue.
4. I have to spit up a lot of thick phlegm every morning.
5. I have a lot of thick mucus in my throat, which causes me to gag, and sometimes even vomit when I try to remove it.
6. My throat gets awfully dry.
7. My mouth gets awfully dry; or, my mouth is so dry that I feel as though it's full of cotton.
8. Every morning when I first awaken I have a sore throat which passes off after I am up and about for a while.
9. I have a burning pain in the tongue; or, my tongue gets awfully sore and sensitive at times.
10. I have a metallic taste in my mouth.
11. I have a lump in my throat; or, the muscles of my throat seem to get tired easily; or, I have pain in my throat when I swallow; or, there is a painful spot inside my throat.

C. LARYNGEAL SYMPTOMS:

1. I have to clear my throat constantly.
2. I feel as though there is something down in my throat, but I can't seem to get it up; if I could, I think that I would feel better.
3. I get hoarse off and on, even though I don't have a cold.

D. CHEST SYMPTOMS:

1. Cough.
2. I often get a burning sensation in the middle of my chest when I take a deep breath.
3. I hear a wheezing in my chest, particularly when I lie down at night.
4. I seem to get short-winded very fast; I must have something wrong with my lungs or my heart.

TABLE II

SOME CAUSES OF INCREASED VISCOSITY OF THE MUCUS AND VARYING DEGREES OF DRYNESS OF THE MUCOUS MEMBRANE OF THE UPPER RESPIRATORY TRACT

Etiologic Agent	*Mechanism Postulated*
1 Vitamin A deficiency	1 Epithelial change
2 Nicotinic acid deficiency	2 Epithelial change
3 High carbohydrate intake	3 Increased mucin content
4 Fear and anxiety	4 Vasoconstriction due to increased sympathetic tone
5 Smoking	5 Same due to nicotine
6 Excessive coffee drinking	6 Same due to caffeine, dehydration caused by diuretic effect of caffeine
7 Chilling of the body surface	7 Reflex vasoconstriction
8 Ingestion of iced beverages	8 Reflex vasoconstriction
9 Post menopausal (natural or surgical)	9 Loss of trophic effect of sex hormones on mucosa
10 Diabetes mellitus	10 Same as No 3
11 Hypothyroidism	11 Increased sensitivity to endogenous epinephrine
12 Hyperthyroidism	12 Increased tissue metabolism
13 Excessive perspiration	13 Dehydration
14 Diarrhea	14 Dehydration
15 Vomiting	15 Dehydration
16 Fever	16 Dehydration
17 Post alcohol ingestion	17 Dehydration
18 Diabetes insipidus	18 Dehydration
19 Nephrotic syndrome	19 Dehydration
20 Insufficient intake of water	20 Insufficient hydration
21 Acute and chronic infection	21 Change in membrane
22 Belladonna or other parasympathetic blocking agents	22 Removes parasympathetic antagonism and leaves sympathetics unopposed
23 Diuretics	23 Dehydration
24 Ephedrine amphetamine benzedrine, and other sympathomimetic drugs	24 Sympathetic preponderance
25 Antihistaminic drugs	25 Parasympathomimetic action; abolishes antagonistic action of histamine to epinephrine
26 Oxygen inhalation	26 Dehydration
27 Anatomic abnormalities of the external nose, septum, turbinates, nasopharynx new growths, foreign bodies	27 Cause abnormal negative pressure, air streams interfere with ciliary activity, stagnation of secretions
28 Surgical removal of important structures	28 Removal of mucus producing structures
29 Mild allergic states with any of the factors named herein superimposed	29 Decreases water content of mucus
30 Lowered atmospheric humidity	30 Decreases water content of mucus
31 Central heat without proper humidifying apparatus	31 Decreases water content of mucus
32 Air conditioning	32 Reflex vasoconstriction
33 Inhalation of drying or astringent dusts	33 Drying of mucus

ETIOLOGY.

Some of the causes of increased viscosity of the mucus and varying degrees of dryness of the mucous membrane of the upper respiratory tract are listed in Table II.

PHYSICAL FINDINGS.

The color of the mucous membranes varies from an angry, dark red to a very pale pink, the so-called normal color range being seen at times.

There may be an appearance of normal moisture, or the mucosa may be so dry and the light reflex so dull that one almost expects to see cracks in it. Sometimes small crusts of dried mucus will be found on such membranes.

In some instances, no evidence of mucus is present other than the impression gained from the glistening appearance of the membrane; however, when a cotton-tipped applicator is lightly passed over the mucosa, a thin layer of very viscid mucus will be observed. The surface tension of this material is so great that it is often very difficult to remove it for transfer to a microscope slide for examination.

At times, mucus threads joining adjacent structures will be encountered. Small clumps of globules are frequently found. If these occur on the vocal cords, they may be confused with small polyps or nodules, but, of course, they will disappear after the patient has coughed several times. Large accumulations of mucus will be found in the presence of anatomic obstruction.

The mucus is usually clear in color but becomes more opaque as the viscosity increases. When it has been present on the membrane for a prolonged period, it may become somewhat yellow in tint. If the condition is due to excessive smoking, it may be stained by the nicotine and tars from the tobacco. At times, it may be blood-streaked, particularly after nasopharyngeal or laryngeal "hawking".

Just as any shade of membrane color may accompany this condition, so any degree of membrane engorgement may be present. In some instances, the turbinates will shrink normally when a vasoconstrictor is applied, while in others there will be little change. This is some indication of the submucosal tissue changes which have occurred.

CYTOLOGIC FINDINGS.

Cytologic study of the mucus is an indispensable aid in the diagnosis of this condition.

The first characteristic noted is in the preparation of the material for staining. Because of its increased viscosity, one has difficulty spreading it into a thin layer with the usual match stick or wire applicator. Then, a long time is required for drying prior to fixation with heat. To speed the process, we sometimes direct a stream of compressed air onto the smear.

During staining with Hansel's stain, decolorization is rather difficult, and frequently one finds it necessary to apply alcohol several times before a sufficient amount of methylene blue has been removed. Even after extensive decolorization royal blue will be the predominant color when the stained smear is held up to be examined by daylight.

Upon examination of the stained smear, several findings are noteworthy. There is usually a relatively large amount of homogeneous-looking mucus containing few cellular elements; however, small accumulations of leucocytes and/or eosinophiles may be found in varying proportions in certain areas, and it is not unusual to see a surprising number of nonlobulated leucocytes; this latter phenomenon we have considered an indication of chronicity. The thicker the mucus, the more epithelial cells will be found in the smear. Quite often one will encounter a great many bacteria. This may indicate either a subacute infectious process or stagnation of secretions.

TREATMENT.

In a recent consideration of one aspect of this problem[1], a rather dismal outlook was given as far as therapy was concerned. We are not in agreement with this hopeless attitude, and feel that otolaryngologists have much to offer these patients.

Definitive treatment is most to be desired, but until this can be accomplished, or if it cannot be achieved, the patient should be given the benefit of symptomatic relief. We shall consider only symptomatic therapy in this presentation.

Symptomatic treatment may be directed towards any one or more of the following:

1. Moistening the mucous membranes.

2. Liquifying the viscid mucus.

3. Stimulating mucus production by systemic administration of drugs.

4. Stimulating mucus production by local application of drugs.

The efficacy of treatment will depend largely upon the amount of metaplastic change which has occurred in the mucosa.

1. Methods of moistening the mucous membranes. These range from the use of a steam kettle to moisten the inspired air to douching the nose with a saline solution several times daily. Patients also use oils and petrolatum to achieve this effect. Some individuals use the common vasoconstrictor preparations for this purpose, although they erroneously believe them to be effective because they shrink the mucous membranes. These methods are not satisfactory, because of the short-lived relief of the symptoms that they provide.

2. Methods used to liquify viscid mucus. Anything that moistens the membranes will, to some extent, also liquify viscid mucus; however, certain mucolytic agents are available. These include: five per cent aqueous solution of ammonium chloride, Alevaire (R), a saturated solution of potassium iodide given orally, and intravenous sodium iodide.

3. Mucus-stimulating materials administered systemically. In addition to the aforementioned iodides, any adrenergic blocking agent may be used. We have used Hydergine (R) to great advantage in this connection[2,3]. The dosage is one sublingual tablet four to six times daily. At times, it is necessary to reinforce the action of Hydergine (R) by the concomitant administration of 15 grains of prostigmine bromide. One may take advantage of the side actions of Rauwolfia serpentina or chlorpromazine to stimulate production of turbinate engorgement and mucus production. All of these agents will give a certain degree of central sedation.

4. *Mucus-stimulating materials applied locally.* Proetz[4] recommends a solution of five per cent ethyl alcohol, three per cent glycerine, and 0.9 per cent sodium chloride in distilled water to stimulate the nasal glands and to retain moisture. The use of 0.25 cc of a 1:2000 solution of prostigmine methylsulfate sprayed into each nostril four times a day has been advocated by Soskin and Bernheimer.[5] Glas[6] employed histamine iontophoresis in increasing doses, beginning with a solution of 1:2000 and reported excellent results. Van Dellen, Bruger and Wright[7] used strong solutions (25 per cent) of acetyl-beta-methyl-choline with some success. We have used nasal sprays and drops of histamine in dilutions varying from 1:2500 to 1:5000 in those cases without too-far advanced metaplasia. The solution can also be used in the form of Dowling packs in the office. Their application results in engorgement of the turbinates and an increased flow of mucus of thin consistency.

If the patient is allergic, an attack of sneezing will be precipitated when the solution is introduced into the nose. It is rather dramatic to use such preparations in wide-open nasal passages which the patient believes are blocked. Within a few minutes there is a diminution in the volume of the lumen, yet the patient states that he feels a great deal better.

DISCUSSION.

It has been said[7] that "careful investigations by able workers have left the cause of most nasal troubles still in doubt." This is not entirely correct. What is true is the fact that much of the basic work has been ignored, and our thinking has been too heavily oriented in terms of hypersecretion as a manifestation of infection, allergy, or the need for surgical intervention. In short, we have been too prone to think in terms of overfunction of the respiratory mucosa as a cause of symptom production. Scant attention has been given to "underfunction" as a basis of symptoms. Undoubtedly, we have been abetted in this error by the fact that patients have a tendency to be etymologically sloppy in their selection of words to describe symptoms. As an example, they may often use the word "blockage" when "stuffiness" would be more accurate.

SUMMARY.

1. A number of symptoms commonly encountered in the practice of otolaryngology are considered in terms of increased viscosity of mucus, and varying degrees of dryness of the mucous membranes of the upper respiratory tract.

2. Treatment based on this concept is discussed.

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705 Baronne Building.

CONGENITAL PERICHONDRITIS ASSOCIATED WITH ACHONDROPLASTIC.*

ROBERT C. KRATZ, M.D.,

Newport, Ky.

At the Cincinnati General Hospital I recently had the opportunity to examine and treat two cases of perichondritis auricularis, which, in my opinion, were congenital. Since I can find nothing in the literature on congenital perichondritis auricularis, these two cases are reported here.

An achondroplastic dwarf was born March 3, 1954, at the Cincinnati General Hospital. This child was the sixth offspring of normal parents. The other five siblings were all healthy and normal. On May 13, 1955, 14 months later, a second achondroplastic dwarf was born to the same parents. These two siblings had identical skeletal deformities, but the second child had a severe cleft palate, while the first had only a bifid uvula. Both of these infants developed bilateral serous perichondritis auricularis. The older of the two developed the condition in the fourth week of life, while the younger infant developed the effusion during the second week. These two children were treated differently and with varying degrees of success.

Case 1. The older child was treated by aspiration of a clear yellow fluid and the application of a collodion cotton mold, which was removed after six days. Three days later, however, the child was returned because the effusion recurred. Again a cotton collodion mold was applied, and not removed for 15 days. Following the second removal there was no recurrence, and the result was excellent. The duration of the disease by this treatment was 41 days.

The younger child was treated by repeated aspirations of the fluid and applications of a wet cotton mold. These aspirations were repeated every three days, and a wet cotton mold

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* From the Department of Otolaryngology at the University of Cincinnati.

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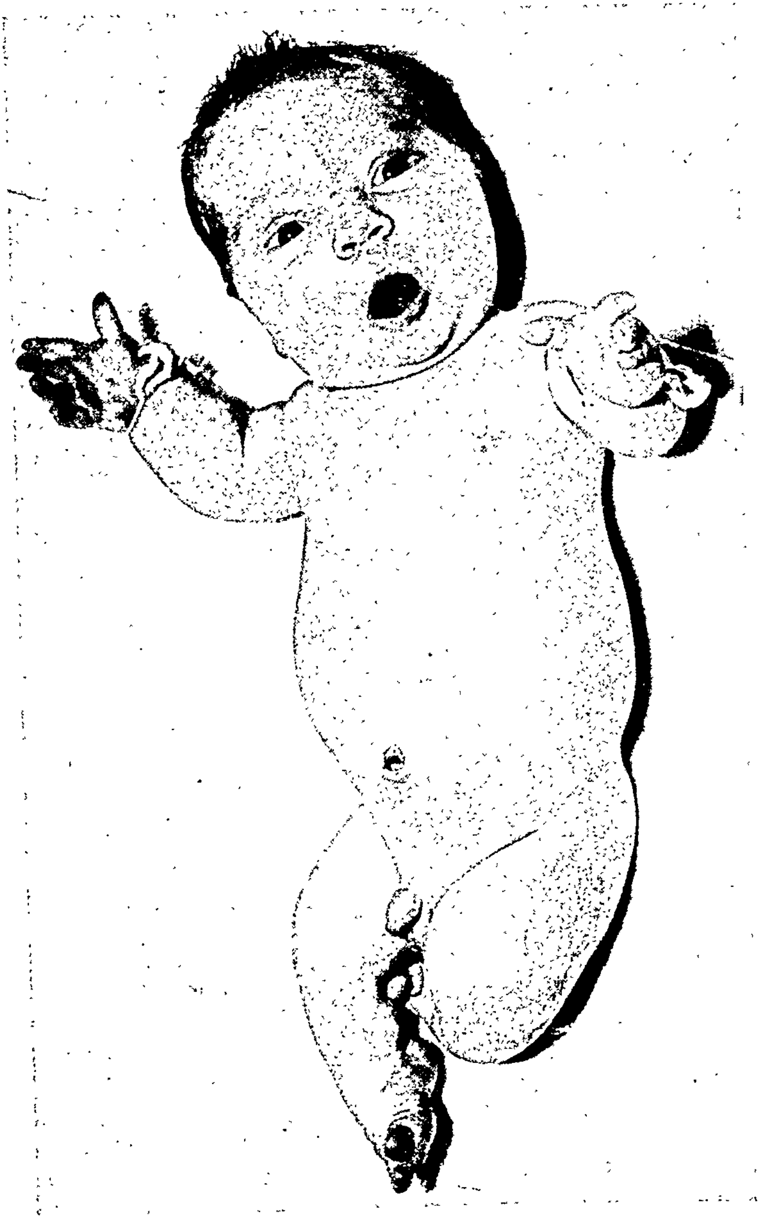

Fig. 1, Case 2. The second and youngest of the two achrondoplastic infants. This photo was made at one week of age. The ears were normal when this photo was taken.

Fig 2 These photos illustrate the right and left ears of the second of the two siblings at the age of five weeks The preceding siblings had the same appearance at approximately the same age

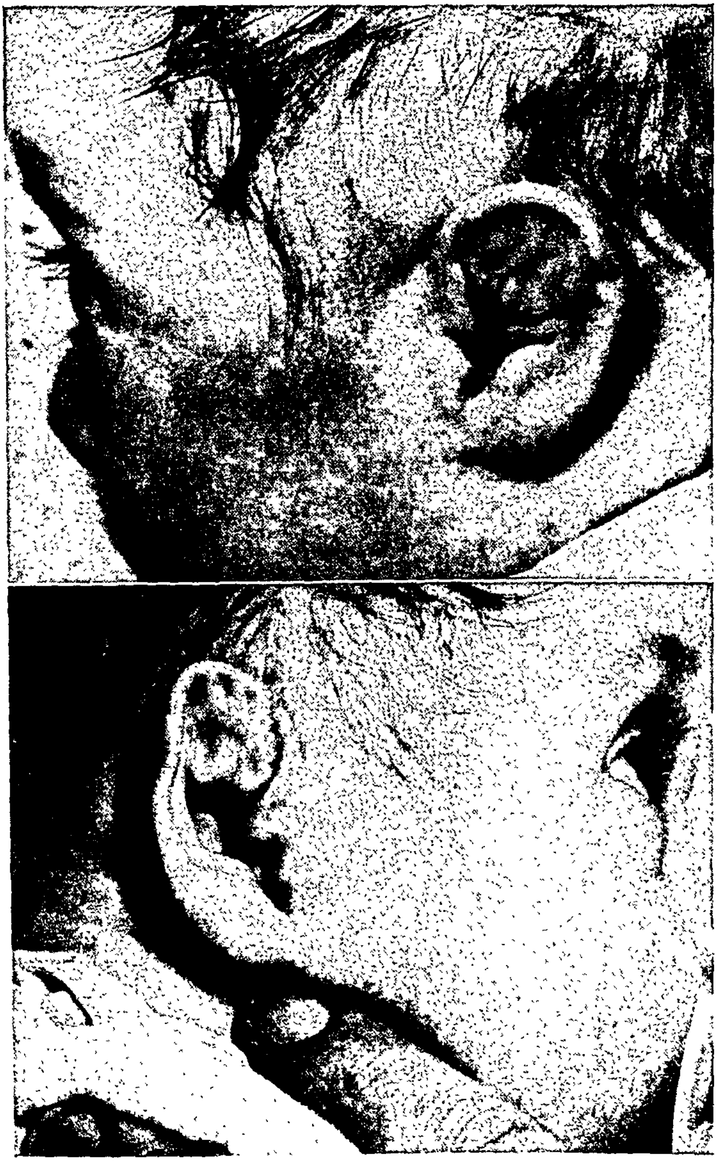

Fig. 3. The second of the two siblings was treated with wet cotton molds. The above photos illustrate the poor result from this treatment. The above photos were taken at the age of two months. The older sibling, who was treated with an ear mold, had an almost perfect result in contrast to the pictures.

reapplied. After two-and-one-half weeks of treatment the condition had not improved. The swelling extended to the place of closing the external auditory meatus. A cotton collodion mold was then applied. This was changed three times before the condition subsided. The result was guardedly fair. The ears were thickened and slightly deformed. The duration of the disease was 68 days.

Discussion:

Since 1941 there have been four achondroplastic dwarfs born at the Cincinnati General Hospital, including the two just mentioned. It could not be determined whether the other two children developed perichondritis auricularis, but since there is no mention made in their charts of the condition I assume they did not.

Judging from my limited experience with two cases of this rare condition, it would seem that the treatment of choice is to aspirate the fluid and fit an ear mold, which is held in place by an elastic type bandage, and is not removed until it becomes loose. It would seem also that treatment should be carried out for a month or longer.

SUMMARY.

Two cases of presumed congenital perichondritis auricularis occurring in achondroplastic siblings are presented.

Suggestions for the treatment of congenital perichondritis auricularis have been offered.

Third and Washington Streets.

In Memoriam

JOHN MACKENZIE BROWN, M.D.

1878 - 1955

John Mackenzie Brown was born in London, Ontario. Canada, in 1878. He received his preliminary education and was graduated from medical school at the University of Western Ontario in 1899. Shortly thereafter, he decided to come to the Southwest and made his permanent home in Los Angeles at the turn of the century, becoming an American citizen within a few years. Becoming interested in otolaryngology early in his career, he decided to obtain special training in this field and traveled extensively studying at the University of Michigan, University of Pennsylvania, Harvard, Edinburgh, and at several European centers.

His skill in his special field was quickly recognized, and he was appointed to a number of hospital staffs in Los Angeles. He was one of the first members of the staff at Children's Hospital, and remained active in that institution from 1909 until 1955. Not only did he serve as Chief of the Department of Otolaryngology, but also served as Chief of Staff at that hospital for a number of years. He maintained affiliations with many other hospitals in the city, including St. Vincents Hospital, the Eye and Ear Hospital, Hospital of the Good Samaritan, and the Cedars of Lebanon Hospital. His services as a consultant were constantly sought at these and other hospitals.

With the re-opening of the School of Medicine at the University of Southern California in 1928, Doctor Brown became vitally interested in the welfare of this institution and became Professor and Head of the Department of Otolaryngology in 1930. Several years later he became Chief of the Department of Otolaryngology at the Los Angeles County General Hospital, and even after his promotion to Professor Emeritus in 1950, he maintained an active interest in the work of both institutions.

The Residency Training Program and the Postgraduate Education in Otolaryngology were greatly stimulated and put

JOHN MACKENZIE BROWN, M D

1878 - 1955

on a solid footing during his period of leadership. His Wednesday rounds at 1:00 o'clock became traditional at the County Hospital and were renowned throughout the country for their excellence and stimulation.

Doctor Brown was deeply interested in the national otolaryngological societes, and served as an officer in many of them. In addition to serving as President of the American Laryngological, Rhinological and Otological Society, and President of the American Academy of Ophthalmology and Otolaryngology, he was also a member of the Los Angeles Society of Ophthalmology and Otolaryngology, the Pacific Coast Oto-ophthalmological Society, the American Otological Society, and the American Laryngological Association. He was a devoted member of all of these organizations and rarely failed to attend a national or local meeting of any society in the field of otolaryngology.

During his many years in the practice of otolaryngology, he contributed much to the advancement of the specialty and his contributions in several fields are world famous. His work on the frontal sinus, including his advocacy of early trephine in acute frontal sinusitis and conservative surgery in chronic frontal sinusitis; his work in branchial-cleft anomalies and his interest in intracranial complications were well recognized throughout the world. He served as Associate Editor of the Archives of Otolaryngology for many years.

All of these sterling qualities and great accomplishments in the field of medicine do not tell the real story of "J.M.", a truly beloved physician. He will be remembered by many thousands of patients who knew him as a kind and able physician with real devotion to the sick. To his fellow physicians, he was known as an astute diagnostician and a very able surgeon in the field of otolaryngology. To many otolaryngologists now practicing, not only on the West Coast but also in various other parts of the country, Doctor Brown will always be the great teacher, the great educator, and the kindly advisor who guided and helped innumerable young men in the specialty of otolaryngology. He was deeply interested in people and in education. He was a strong guiding force in the early days of organization of the School of Medicine at the University of Southern California, and for many years was a member of the executive committee of the faculty. A large group of his former students and friends have for years met at the annual meetings of the American Academy of Ophthalmology and Otolaryngology as the "John Mackenzie Brown Alumni Group", and have awarded an annual recognition to that member of the group who made the best contribution to the literature in that year. This award is considered a great honor. In 1949, a testimonial dinner was given to Doctor Brown by a large number of otolaryngologists in the Southern California area upon the occasion of his completion of fifty years of practice. At this testimonial dinner, an oil painting was presented to him by his devoted friends, and copies of this painting hang in many otolaryngological offices on the West Coast.

"J.M." was a most humble and modest person who always tried to remain quietly in the background; nevertheless, his great character and human qualities were so evident that many honors were presented to him during his years in practice.

Remaining actively engaged in practice and in his educational work until the very end, Doctor Brown passed away in his sleep on December 31, 1955. He is survived by his widow, Marian, two daughters, two sons, and six grandchildren.

The John Mackenzie Brown Memorial Fund has been established to perpetuate the name of the man who was known as the dean of otolaryngologists on the West Coast, and who made contributions to the specialty, which will be permanent in the archives of otolaryngology. This fund will be used to build a lecture hall in the new University of Southern California Medical School Center. This will be a fitting memorial to Doctor Brown, who was very anxious to see education in medicine raised to the highest possible level.

Contributions to the University of Southern California Medical School, attention John Mackenzie Brown Memorial Fund, may be forwarded to the fund office, Suite 308, 1136 West Sixth Street, Los Angeles 17, California. H. H.

NATIONAL SOCIETY MEETINGS.

Schedule of Meetings for 1956:

American Board of Otolaryngology, to be held at the Sheraton-Mt. Royal, Montreal, Canada, May 6-11.

American Otological Society, Inc., to be held at the Seigniory Club, Ottawa, Canada, May 11-12.

American Laryngological Association to be held at the Seigniory Club, Ottawa, Canada, May 13-14.

American Broncho-Esophagological Association, to be held at the Sheraton-Mt. Royal, Montreal, Canada, May 15-16, (afternoons).

The American Laryngological, Rhinological and Otological Society, Inc., will hold its Annual Meeting at the Sheraton-Mt. Royal, Montreal, Canada, May 15-16-17 (mornings only).

Please make early plans to attend the 1956 Spring Meetings in Canada. Both the Seigniory Club and Montreal present most attractive features for you and your family. More information about the places will be given later.

Reservations at the Sheraton-Mt. Royal Hotel should be made early by addressing the Reservation Supervisor, 1455 Peel Street, Montreal, P. Q., Canada.

WASHINGTON UNIVERSITY, SAINT LOUIS, DEPARTMENT OF OTOLARYNGOLOGY.

Offers a Basic Science Course in Otolaryngology to start Monday, September 17, 1956. Complete information about the course may be obtained by writing to Theo. E. Walsh, M.D., Head of the Department of Otolaryngology, 640 S. Kingshighway, St. Louis 10, Missouri.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

The Sixth International Congress of Otolaryngology will take place in Washington, D. C., from Sunday, May 5, through Friday, May 10, 1957, under the presidency of Arthur W. Proetz, M.D.

The selected subjects for the Plenary (Combined) Sessions to be held Monday, Wednesday and Friday mornings will be:

1. Chronic Suppuration of the Temporal Bone.
2. Collagen Disorders of the Respiratory Tract.
3. Papilloma of the Larynx.

Outstanding internationally recognized authorities will open the discussion of each of these subjects.

Two types of communications are invited: *1.* Contributions to the discussions of the selected subjects, limited to five minutes. *2.* Original papers, limited to 15 minutes. These should be in one of the four official languages: English, French, German, Spanish.

Motion picture films will be shown continuously except during the Plenary Sessions. There will be both scientific and technical exhibits. Those wishing to submit contributions to the program should communicate with the General Secretary.

Announcement of the Congress has been sent to all otolaryngologists whose names and addresses could be obtained. Additional details concerning registration, housing, entertainment, etc., will be sent to those who have indicated to the General Secretary that they wish further information.

The subscription for Members (physicians) is $25.00 U.S.A. This includes the privilege of attendance at all official Congress meetings except the banquet for which an additional charge will be made. Other persons accompanying Members may be registered as Associates at a fee of $10.00 U.S.A.

An interesting program of social functions, visits to points of interest in and around Washington and post-Congress tours is being arranged. The American Express Company is the official travel agent for the Congress. Their offices throughout the world are available for travel arrangements to the Congress and for post-Congress tours.

All communications should be addressed to the General Secretary, Paul H. Holinger, M.D., 700 N. Michigan Ave., Chicago, Ill., U.S.A.

INDIANA UNIVERSITY MEDICAL CENTER.

The Department of Otolaryngology, Indiana University School of Medicine, offers its annual Anatomical and Clinical Course in Otolaryngology March 26th to April 7th, 1956.

Applicants should address The Post-Graduate Office, Indiana University Medical Center, Indianapolis 7, Indiana.

MOUNT SINAI HOSPITAL

Mount Sinai Hospital offers three post graduate courses given in affiliation with Columbia University. The course on Indirect Laryngoscopy is scheduled for Feb. 13-14, 1956; a course on Voice and Speech Therapy will be given Feb. 15-16, 1956 and the course in Audiology Feb. 17-18, 1956.

These courses may be taken separately or as one combined course.

For further information address The Registrar for Post Graduate Medical Instruction, Mount Sinai Hospital, Fifth Avenue and One-Hundredth Street, New York (29), N. Y.

A special course in Endaural Otologic Surgery will be given by Northwestern University February 28 through March 26, 1956. Course is limited to eight otolaryngologists. Instruction to include lectures, cadaver dissection, etc. For further information write Dr. Geo. E. Shambaugh, Jr., Dept. of Otolaryn., Northwestern Medical School, 303 East Chicago Ave., Chicago (11), Ill.

POSTGRADUATE COURSE.

The Mount Sinai Hospital, New York, in affiliation with Columbia University, announces an intensive postgraduate course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty, given by Irving B. Goldman, M.D., and staff, July 14-18, 1956.

Candidates should apply to Registrar for Postgraduate Medical Instruction, Mount Sinai Hospital, 5th Avenue and 100th Street, New York 29, New York.

UNIVERSITY OF ILLINOIS COLLEGE OF MEDICINE.

The next Laryngology and Bronchoesophagology Course to be given by the University of Illinois, College of Medicine, is scheduled for the period March 5 through March 17, 1956. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois, College of Medicine, 1853 W. Polk Street, Chicago 12, Illinois.

POSTGRADUATE COURSE IN OTOLARYNGOLOGY.

The Department of Postgraduate Medicine of the University of Michigan Medical School announces the Otolaryngology Conference to be given at the University Hospital, Ann Arbor, Michigan, on April 19, 20 and 21, 1956, under the direction of Dr. A. C. Furstenberg, Chairman of the Department of Otolaryngology at the University of Michigan Medical School.

Guest lecturers of national prominence, together with members of the staff of the Department of Otolaryngology, will participate in the program.

For further information, write to Dr. John M. Sheldon, Director, Department of Postgraduate Medicine, University Hospital, Ann Arbor, Michigan.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Wm. J. McNally, 1509 Sherbrooke St., West Montreal 25, Canada.
Vice-President: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Secretary-Treasurer: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Seigniory Club, Ottawa, Canada, May, 1956.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Bernard J. McMahon, 8230 Forsyth Blvd., Clayton 24, Mo.
First Vice-President: Robert L. Goodale, 330 Dartmouth St., Boston, Mass.
Second Vice-President: Paul H. Holinger, 700 North Michigan Ave., Chicago 11, Ill.
Secretary: Harry P. Schenck, 326 South 19th St., Philadelphia 3, Pa.
Treasurer: Fred W. Nixon, 1027 Rose Building, Cleveland, Ohio.
Librarian, Historian and Editor: Edwin N. Broyles, 1100 North Charles St., Baltimore, Md.
Meeting: Mount Royal Hotel, Montreal, Canada, May, 1956.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Dean M. Lierle, Iowa City, Iowa.
President-Elect: Dr. Percy Ireland, Toronto, Canada.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Mount Royal Hotel, Montreal, Canada, May, 1956.

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Vice-Chairman: James W. McLaurin, M.D., Baton Rouge, La
Secretary: Hugh A. Kuhn, M.D., Hammond, Ind.
Representative to Scientific Exhibit: Walter Heck, M.D., San Francisco, Calif.
Section Delegate: Gordon Harkness, M.D., Davenport, Iowa.
Alternate Delegate: Dean Lierle, M.D., Iowa City, Iowa

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Daniel S. Cunning, 115 East 65th St., New York 21, N. Y.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia, Pa.
Meeting: Sheraton Mount Royal Hotel, Montreal, Canada, May 15-16, 1956 (afternoons only).

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill., October, 1956.

THE AMERICAN RHINOLOGIC SOCIETY

President Dr Ralph H Riggs, 1513 Line Ave, Shreveport, La
Secretary Dr James Chessen, 1829 High St, Denver, Colo
Annual Clinical Session Illinois Masonic Hospital Chicago Illinois October, 1956
Annual Meeting Palmer House, Chicago, Illinois, October 1956

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President Dr D M Lierle, University Hospital Iowa City Iowa
Secretary Treasurer Dr Michael H Barone, 468 Delaware Ave, Buffalo 2, N. Y.
Meeting Palmer House, Chicago, Ill, October 1956

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

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EXFOLIATIVE CYTOLOGY OF THE LARYNX —A PRELIMINARY REPORT.*

CLAY W. WHITAKER, M.D. (by Invitation),
and
EDWARD E. SIEGLER, M.D. (by Invitation),
Cleveland, Ohio

We have been investigating the possible practical value of exfoliative cytology of the larynx as an aid in early diagnosis of carcinoma.

It is a well known fact that laryngeal carcinomas sometimes are delayed in diagnosis. There may be a considerable period between the time the patient first sees a physician until the diagnosis is final The doctor may use the approach of watchful waiting rather than alarm the patient by immediate hospital admission for formal laryngoscopy and biopsy. We hope that exfoliative cytology can shorten the delay period by giving the office practitioner a relatively easy and atraumatic method for screening suspected lesions of the larynx.

The technique used in this study is to cocainize the larynx as one would for a direct laryngoscopy and then with mirror laryngoscopy to obtain a direct smear, using the Wagner's camel's-hair brush and wiping it firmly over the suspected lesion (see Fig. 1). This brush is obtainable as a standard

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item from surgical supply houses. In taking smears from growths of the vocal cords the best results have been obtained by placing the brush between the cords, having the patient phonate, and moving the brush from anterior to posterior with the cords approximated. In extrinsic lesions it is necessary to rub the brush firmly over the lesion.

As soon as the specimen is obtained the material is painted on a labeled glass slide, and the slide is immediately dropped into a fixative solution composed of equal parts of 95 per cent alcohol and ether. The slide must remain in this solution at least two hours, although it may remain as long as one week After fixation the slide is ready for staining and final processing. Processing is the same as for Papanicolaou smears obtained from any other organ, using the OG-6, EA-50 stain.

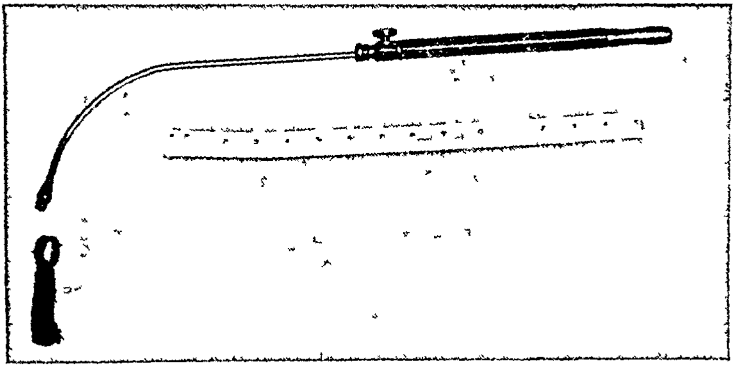

Fig 1

In our study the slides were screened by a technician who knew the clinical diagnosis but not the biopsy diagnosis All positive, suspicious, and questionably negative cases were examined by the cytologist, who also had no knowledge of the biopsy diagnosis.

In the first 18 months of this study 99 cases were examined. In 95 of these the cytological interpretation was checked by biopsy diagnosis. The other four cases were all patients who had laryngoscopies performed for reasons other than biopsy, usually paralyzed vocal cords

TABLE I LARYNGEAL SMEAR STUDY—BIOPSY DIAGNOSES

Laryngeal Nodule	30
Squamous Cell Carcinoma	23
Papilloma	11
Chronic Laryngitis	10
Keratosis	8
Leukoplakia	6
Granuloma	2
Cyst	2
Negative	3
	95

Table I shows the results of biopsies on the group of 95 patients Of these, 72 had benign lesions and 23 had squamous cell carcinomas

TABLE II LARYNGEAL SMEAR STUDY—CLINICAL DIAGNOSES

Laryngeal Nodule	27
?? Carcinoma	21
Carcinoma	17
Laryngitis	13
Polyps	8
Leukoplakia	6
Papilloma	6
Cyst	1
	99

The next table shows the clinical diagnosis in this same group of patients Here 38 were classed as either apparent or suspected carcinoma Of the 23 proved carcinomas, 22 were included in one of these two groups The suspected group was usually labeled "rule out malignancy" The large number in this class indicates to some extent the difficulty in making a clinical diagnosis of early carcinoma of the larynx.

Interpretation of laryngeal smears from the 23 proved carcinomas gave 15 positive, four suspicious, and four negative, an apparent accuracy of 65 per cent. In practice it would obviously be wise to class the suspicious as at least tentative positives until biopsy can confirm the results. On this basis, the useful accuracy would be over 80 per cent.

The remaining four cases, false negatives, are not unexpected. The inherent histological characteristics of the larynx make it highly improbable that 100 per cent accuracy of diagnosis will ever be achieved—even biopsy sometimes fails. In addition, we found in the early days of our study that the examiner occasionally did not obtain the specimen from the suspected lesion. For example, one patient had an early lesion on the laryngeal surface of the epiglottis difficult to visualize. A smear taken by a first-year resident was negative; one taken at the same examination by a third-year resident was positive.

Probably the most important reason for false negatives is that it is not uncommon for carcinoma of the larynx to invade before a typical carcinoma *in situ* stage is found on the exfoliating surface. Carcinoma *in situ* is one in which the malignant cells involve the surface but have not invaded beyond the basement membrane; furthermore, some tumors are very well differentiated, and the exfoliating cells are so slightly altered that they are not distinguishable from the normal or very slightly abnormal cells frequently seen in benign conditions.

It is encouraging to find that there were no false positives in the entire group. There were, however, six cases reported as suspicious from the 72 negative biopsies. We think that these false suspicious reports may be occasioned by abnormalities of the surface epithelium, such as atrophy, hyperplasia. acanthosis, hyperkeratosis, or dyskeratosis. In any event all cases either positive or suspicious should be confirmed by biopsy. The cytological test is intended to be a screening procedure for biopsy rather than an ultimate diagnostic method.

The following three case histories have been summarized to help show the possible future value of this procedure

The first patient a 29 year old white female, was first seen at our hospital 11 years ago in 1944, when she was only 18 years of age At that time she had lost her voice, and a lesion of the larynx was biopsied and interpreted as a papilloma On review of this slide we now classify this lesion as a carcinoma *in situ* Her symptoms recurred in 1950 and a biopsy was again interpreted as a papilloma and again on subsequent review of the biopsy we have also reclassified this lesion as carcinoma *in situ* Her admission this year was due to two months of hoarseness Laryngeal brush smears were positive for tumor cells but this time the biopsy showed definite microscopic evidence of shallow invasion in addition to the surface involvement Total laryngectomy was performed

This case illustrates the value of detection of a carcinoma which should have an extremely favorable prognosis

The other interesting aspect of this case is the 11-year interval between the initial biopsy which showed no invasion and the recent biopsy which did show evidence of shallow invasion Clerf has reported one case similar to ours, of an 11-year interval, and Stewart reported one of a seven-year interval.

The second patient a 57 year old white male was followed for six months by an otolaryngologist in another city In this case complaint was hoarseness and the lesion of the larynx was interpreted as an inflammatory lesion by the original examiner He made many sets of smears and cultures for acid fast bacilli The patient was referred to an otolaryngologist at St Luke's Hospital who made a clinical diagnosis of carcinoma Laryngeal brush smears were positive for tumor cells and the biopsy showed a squamous-cell carcinoma

Here is an example of a six-month delay due to an error in clinical interpretation Had the original physician had a simple office procedure available, he might have been alerted sooner.

The third patient a 56 year old white male entered the hospital with the complaint of hoarseness of one year's duration Five months previously, a smaller tumor had been removed from one of his vocal cords at another hospital This was interpreted as negative for tumor His hoarseness continued The clinical diagnosis on this admission was probable laryngeal nodule but the possibility of carcinoma could not be completely excluded The tumor was described as small and on the under surface of the vocal cord The smears were strongly positive The biopsy was diagnosed as squamous cell carcinoma

This is an example of a case in which the cytology was so strongly positive we would have asked for the biopsy despite the clinical appearance of the lesion or the history

From these preliminary results it appears that laryngeal cytology may have particular advantages in diagnosis. Because of speed and simplicity it could be a quick office test for lesions which are clinically doubtful in nature. It could, incidentally, also do what cervical cytology has done in encouraging the physician to visualize the organ under study. Most important, because of its atraumatic nature it could be used as a serial test to follow suspicious conditions or biopsy-proved cases which have been treated by irradiation alone. In the latter, a recurrence might be detectable earlier cytologically than clinically. Another application might be in those patients whose biopsy findings were inconclusive. At present these patients require close following with frequent biopsies, an uncomfortable and expensive program. A program of frequent office visits with brush specimens taken on each visit would be less expensive for the patient and would enable the physician to add to the clinical evaluation a simple aid in follow-up care. Needless to say, if the clinical appearance of the larynx were noticeably different from previous examinations a biopsy would be in order.

This first series has included only a selected group of hospital patients. We cannot yet predict that the results will be similar with a wider range of subjects. The work is being continued to include office patients, and those who on the basis of clinical examination are not to be biopsied at the same time. Some of these patients will have such premalignant conditions as leukoplakia or atypical keratosis, and may develop a carcinoma *in situ*.

Since carcinoma *in situ* can be expected to exfoliate diagnostic cells at least in some instances, and since less than 2 per cent of laryngeal carcinomas are now diagnosed in this non-invasive stage, any additional tool to screen symptomatic patients before the development of a late lesion would be worthwhile. If, in even a small percentage of these, unsuspected positives can be detected and confirmed, the procedure will prove its value.

EFFECTIVENESS OF CONDITIONED ELECTRODERMAL RESPONSES (EDR) IN MEASURING PURE-TONE THRESHOLDS IN CASES OF NON-ORGANIC HEARING LOSS.

ROBERT GOLDSTEIN, Ph D.,
St. Louis, Mo.

The problem of non-organic hearing loss grew to major proportions for the military services during and following World War II, and it has continued to be significant in recent years, especially for the Veterans Administration. Now, with increasing concern in industry about the effects of noise on hearing and subsequent compensation claims for hearing losses, the problem of non-organic hearing loss will undoubtedly become magnified.

Relatively successful tests have been developed for the *detection* of malingering and psychogenic deafness[12,17], but these tests do not usually *measure the threshold of hearing.* For years, otologists and audiologists have looked for an "objective" test for the measurement of pure-tone thresholds, that is, a test in which responses to tone can be made independent of the will or control of the person tested, and in which the responses can be scored with a minimum of subjective interpretation. A test that seemed to hold promise of providing a measure of threshold in a relatively objective manner involved the use of electrodermal responses (EDR)* to tone as an indication of hearing.

The use of changes in the electrical properties of the skin as an indication of hearing in cases of non-organic hearing loss was reported as far back as 1915. Wiersma[44] observed changes in the electrical resistance of the skin associated with auditory stimuli to help him distinguish organic deafness

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* More commonly designated as "psycholgalvanic skin response" or "galvanic skin response" and variously abbreviated PGSR, GSR PGR PG and GR.

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from simulated or from functional deafness. Since that date others have reported[1,11,26,29,33] similar use of electrodermal responses to tone.

Interest in this so-called "objective" test of hearing was revived by Bordley, Hardy and Richter[8] in their report of a modification of the EDR technique. They proposed that for clinical use tones be followed by an annoying shock, so that conditioned responses to tones could be established. This conditioning procedure, then, could make measurement of threshold by the EDR method more feasible, because it would not permit the nervous system of the person tested to adapt to the auditory stimuli even at low sensation levels.

Since the report of Bordley, Hardy and Richter[8], there have been numerous reports[3,6,7,13,14,20,25,31,37] describing the successful use, or the probability of successful use. of conditioned electrodermal responses not only for detecting non-organic hearing losses but also for obtaining accurate audiograms in those same cases. There have also been scattered reports of the failure of the EDR test with some cases of malingering and psychogenic deafness[2,17,18,41].

The purposes of this paper are: (1) to describe the clinical experience at Central Institute for the Deaf with the EDR test as a means of measuring pure-tone thresholds in patients suspected of having non-organic hearing losses; (2) to discuss the problems involved in the EDR test with these kinds of patients.

We are not concerned here with the success of the threat of shock in the EDR test in coercing or encouraging better cooperation in conventional testing procedures. We are considering only the success of the tester in eliciting electrodermal responses to tones so that reliable auditory thresholds can be determined from those responses.

OBSERVATIONS.

Twenty patients suspected of having non-organic hearing losses have been examined by the EDR test. Some of the tests were eminently successful while in other tests the patients gave few or no reliable responses in spite of intensive conditioning. If we take into account the responses during conventional audiometry (patient asked to raise his finger when tone is heard, lower it when tone is no longer heard), as well as the responses during the EDR test, we can describe four general categories of responses during our pure-tone tests.

1. Poor conventional responses—good EDR.

2. Good conventional responses—good EDR.

3. Poor conventional responses—poor EDR.

4. Good conventional responses—poor EDR.

We considered the conventional responses to be poor when they did not occur consistently with a given level of the tone which was apparently supra-threshold, when the pure-tone thresholds were grossly inconsistent with the patient's scores on speech tests or with his obvious ability to hear conversational speech, or when the behavior of the patient indicated that he was deliberately withholding responses.

The 20 cases were about evenly divided among these four categories. The reason for the approximate rather than definite division of cases among the particular categories is the continuous rather than discrete nature of the categories themselves. For instance, those patients whose electrodermal responses were consistent but whose conventional responses were neither very consistent nor very inconsistent could be placed justifiably in either category 1 or 2.

The following examples will illustrate the range of success with EDR as outlined above.

1. POOR CONVENTIONAL RESPONSES—GOOD EDR.

A. M. was a young Air Force nurse with a history of acute aerotitis and an ensuing permanent hearing loss. Malingering was suspected, however, because of inconsistencies in audiometric scores and because of the patient's urgent desire to be separated from military service.

Chart I is a record of the patient's audiogram and speech test results.

from simulated or from functional deafness. Since that date others have reported[1,11,26,29,33] similar use of electrodermal responses to tone.

Interest in this so-called "objective" test of hearing was revived by Bordley, Hardy and Richter[8] in their report of a modification of the EDR technique. They proposed that for clinical use tones be followed by an annoying shock, so that conditioned responses to tones could be established. This conditioning procedure, then, could make measurement of threshold by the EDR method more feasible, because it would not permit the nervous system of the person tested to adapt to the auditory stimuli even at low sensation levels.

Since the report of Bordley, Hardy and Richter[8], there have been numerous reports[3,6,7,13,14,20,25,31,37] describing the successful use, or the probability of successful use. of conditioned electrodermal responses not only for detecting non-organic hearing losses but also for obtaining accurate audiograms in those same cases. There have also been scattered reports of the failure of the EDR test with some cases of malingering and psychogenic deafness[2,17,18,41].

The purposes of this paper are: (1) to describe the clinical experience at Central Institute for the Deaf with the EDR test as a means of measuring pure-tone thresholds in patients suspected of having non-organic hearing losses; (2) to discuss the problems involved in the EDR test with these kinds of patients.

We are not concerned here with the success of the threat of shock in the EDR test in coercing or encouraging better cooperation in conventional testing procedures. We are considering only the success of the tester in eliciting electrodermal responses to tones so that reliable auditory thresholds can be determined from those responses.

OBSERVATIONS.

Twenty patients suspected of having non-organic hearing losses have been examined by the EDR test. Some of the tests were eminently successful while in other tests the patients gave few or no reliable responses in spite of intensive conditioning. If we take into account the responses during conventional audiometry (patient asked to raise his finger when tone is heard, lower it when tone is no longer heard), as well as the responses during the EDR test, we can describe four general categories of responses during our pure-tone tests.

1. Poor conventional responses—good EDR.

2. Good conventional responses—good EDR.

3. Poor conventional responses—poor EDR.

4. Good conventional responses—poor EDR.

We considered the conventional responses to be poor when they did not occur consistently with a given level of the tone which was apparently supra-threshold, when the pure-tone thresholds were grossly inconsistent with the patient's scores on speech tests or with his obvious ability to hear conversational speech, or when the behavior of the patient indicated that he was deliberately withholding responses.

The 20 cases were about evenly divided among these four categories. The reason for the approximate rather than definite division of cases among the particular categories is the continuous rather than discrete nature of the categories themselves. For instance, those patients whose electrodermal responses were consistent but whose conventional responses were neither very consistent nor very inconsistent could be placed justifiably in either category 1 or 2.

The following examples will illustrate the range of success with EDR as outlined above.

1. POOR CONVENTIONAL RESPONSES—GOOD EDR.

A. M. was a young Air Force nurse with a history of acute aerotitis and an ensuing permanent hearing loss. Malingering was suspected, however, because of inconsistencies in audiometric scores and because of the patient's urgent desire to be separated from military service.

Chart I is a record of the patient's audiogram and speech test results.

CHART I.

In this, and succeeding charts, pure-tone and speech thresholds (spondee word lists) determined by standard procedures are given in decibels of hearing loss with respect to normal thresholds. Discrimination scores (PB max.) are given in per cent of words correctly identified from phonetically balanced word lists presented at intensities we'l above the patient's threshold (or estimated threshold) for speech. Pure-tone thresholds determined by the EDR test are given when they are available.

	125	250	500	1000	2000	4000	8000	Speech Thresh.	PB Max.
Right AC	70	60	65	70	75	75	90		88%
BC			40	40	40				
EDR (AC)			10	10					
Left AC	(responses only at maximum output of audiometer)								84%
BC			60	60	60				
EDR (AC)				65					

Electrodermal responses to tone alone were so consistent that no conditioning shocks were used. The patient was in tears after the EDR test had progressed for about 10 minutes. She probably realized that we were not deceived because we presented only tones which were close to normal threshold. The 10 db losses in the right ear at 1000 and 2000 cps represent the patient's maximum hearing losses at those frequencies. We might have obtained consistent responses with weaker tones had we attempted conditioning. The 65 db loss in the left ear at 1000 cps probably represents threshold of hearing in the right ear from sound conducted through the skull. We made no attempt to mask the right ear while testing the left ear.

The other four subjects in this category did not yield electrodermal responses as easily as did A. M., but conditioning of responses was relatively easy in those cases and we considered our judgments of thresholds to be reliable.

2. GOOD CONVENTIONAL RESPONSES—GOOD EDR.

M. H. was a 16-year-old girl who gave consistent and reliable responses during conventional audiometric tests. She was referred for an EDR test because of suspicion of a non-organic hearing loss for the following reasons: *(a)* the examining otologist was able to carry on a direct conversation with her with relative ease in a normal voice in spite of her poor performance during the test of speech discrimination; *(b)* her audiogram, especially for the left ear, was rather unusual: a rising audiogram with maximum sensitivity at 3000 cps is not common in cases of apparent perceptive-type hearing losses; and *(c)* the onset of the hearing loss was rather sudden, apparently only six months prior to visiting the otologist. The cause of the hearing loss could not be ascertained.

The following chart is a summary of the audiometric findings on M. H.

CHART II

	250	500	1000	2000	3000	4000	6000	8000	Speech Thresh	PB Max
Right AC	35	45	45	30	5	15	65	75	67	6%
BC		45	50	30	10	25				
EDR (AC)			50		15	> 40	55?			
Left AC	40	50	50	45	35	20	55	45	71	6%
BC		35	X	X	35	30				
EDR (AC)						45?				

In that the EDR thresholds indicated no more hearing than the conventional audiogram, the conventional audiogram was considered valid, and the hearing loss was regarded as organic

EDR testing was begun at 1000 cps in the right ear. The responses were consistent and the estimated threshold corresponded closely with the threshold determined by standard procedures As the EDR test progressed the responses became less frequent in spite of nearly constant reinforcement Thus, some EDR thresholds given in the chart are qualified as being uncertain At any rate, there were no electrodermal responses to tones at less intensity than the threshold values determined by conventional procedures Of the other four patients in this category, two gave responses as consistent as those of M. H , one gave fewer reliable responses, and one gave more frequent reliable responses

3 POOR CONVENTIONAL RESPONSES—POOR EDR

L S was a 48 year old man who claimed compensation for serious loss of hearing in his left ear as a result of a blow to the head while he was at work

Two EDR tests were given two weeks apart and conventional audiometric tests were done on both days L S cooperated no better during the second attempt at a pure tone audiogram than he did during the first His behavior during the conventional pure tone test was characteristically that of a malingerer By contrast, he seemed to cooperate completely during the speech tests The following chart shows the discrepancy between the pure tone and speech tests No bone conduction tests were attempted There were not enough electrodermal responses to tone to permit an estimate of threshold

CHART III.

	250	500	1000	2000	4000	8000	Speech Thresh.	PB* Max.
Right AC	5	5	10	20	40	30	3	66%
Left AC	35	25	45	50	80	50	10	80%

* Same intensity level for both ears, approximately 45 db above normal speech thresholds.

L. S. did not fail to give clear electrodermal responses to shock. He even gave an occasional clear electrodermal response to live voice close to normal threshold. His responses to pure tones, however, were very sparse; they were less frequent during the second session than they were during the first; nevertheless, during both sessions he admitted, without prompting, that he was hearing many tones. It was almost as though he hoped that his admission would deter us from giving a shock. At no time, however, that he told us he heard a tone did he show what could even be considered as a probable electrodermal response.

We attempted to confirm that L. S. was malingering by the Lombard test, but a white noise at approximately 130 db above 0.0002 dyne/cm^2 produced no detectable effect on his speaking voice. We followed the suggestion of Hanley and Tiffany[22] in trying to disrupt reading and spontaneous speech of L. S. by feeding back his own voice to him at a high intensity and with a delay of about 0.2 second. This procedure had no effect on his speech.

Of the five patients in this group, L. S. gave the poorest electrodermal responses, but none of the others gave enough consistent responses to establish a reliable threshold in either ear at any frequency.

4. GOOD CONVENTIONAL RESPONSES—POOR EDR.

H. H. was a 16-year-old boy who gave a history of rather sudden onset of hearing loss about six months prior to these examinations. His responses during the conventional audiometric tests were consistent, and

CHART IV.

	250	500	1000	2000	3000	4000	6000	8000	Speech Thresh.	PB Max.
Right AC	50	45	55	55	60	45	50	50	58	80%
BC		25	35	45	35	35				
EDR (AC)			50?							
Left AC	40	35	40	30	20	20	35	30	28	90%
BC		15	30	40	20	40				
EDR (AC)				30-40?						

speech thresholds and speech discrimination were commensurate with the pure tone audiogram. The lack of positive otologic findings, however, along with the flighty, distrustful nature of H. H and a story of unhappy home life all led to a suspicion of a psychogenic overlay on a possible organic hearing loss. The test results are summarized in Chart IV.

H. H. was difficult to condition, in spite of almost constant shock reinforcement Shock was definitely annoying and elicited large electrodermal responses A bright floodlight was used later in the test, in addition to shock, as an unconditioned stimulus, but this did not help to condition electrodermal responses to tone The EDR thresholds given in Chart IV are uncertain; more dependence was placed on overt reactions in anticipation of the shock than on the occasional electrodermal responses.

Two of the other patients in this category gave similarly poor electrodermal responses, while two gave a few more dependable responses

DISCUSSION.

We have reported a larger proportion of unsatisfactory results with the EDR test than is generally described in the literature. We are not able at this time to explain the discrepancy; nevertheless, we shall try to account for some of the difficulties which may be encountered in measuring pure-tone thresholds in cases of non-organic hearing loss.

A study on normally hearing children[19] indicates that there may be many completely normal people who are difficult to condition. It is probable, therefore, that ease of conditioning may not be related principally to the nature of the auditory disorder of the person tested, as had been suggested earlier[18], or to the particular conditioning procedure.

It can be inferred from the writings of James[28] and Fuller[15] that relative ease of conditioning in general may be a function of some psychophysiological characteristic of the individual and not of conditioning procedures or of some pathological state. These investigators described differences in conditioning behavior between different breeds of the same species of animals. Lacey, Siegel and Siegel[30], however, looked for some relevant morphological factor in humans, but did not find any relation between body form (height-weight ratio) and either basic skin conduction or electrodermal responses.

The single factor or constellation of factors which characterize humans who are easy to condition or difficult to condition has not been established. Welch[42], Welch and Kubis[43], and

Bitterman and Holtzman[4] did present evidence that anxiety was positively related to success of conditioning. A later study by Bitterman, Holtzman and Barry[5] failed to confirm their index of anxiety as a predictor of the degree of conditioning. Taylor and Spence[40] noted that anxiety neurotics were no easier to condition than other neurotics. Hardy, Wolff and Goodell[23], in their studies on pain, found no relation between apparent emotional tension and magnitude of electrodermal responses during a period of adaptation or habituation. We have not observed that persons who were overtly anxious in the test situation were conditioned any more easily than those who appeared calm.

Taylor and Spence[40] did find that the eyeblink response was conditioned more easily in psychotics than in neurotics, but Greenwald[21] recorded virtually the same EDR from mental patients as he did from normals under the same conditions. Paintal[35] found no impairment in psychotics of the physiological mechanism for producing electrodermal changes, but he did note that psychotics responded far less than normals to the threat of a shock.

Gantt[16], Reese[38], and Reese, Doss and Gantt[39] noted that electrodermal responses as well as other autonomic and skeletal responses were markedly more difficult to condition in psychotics with "diffuse cortical impairment" than they were in normals. Peters and Murphree[36] reported that chronic schizophrenics gave smaller electrodermal responses and were more difficult to condition than normals. They also found that a group of schizophrenic patients who had spent three months learning to solve problems "with insulin stimulated hunger as a motive" reacted more strongly and conditioned more easily than the untreated patients. If, then, we consider some cases of deafness to be symptomatic of a psychosis, we may possibly expect the EDR test to be less successful in those instances than in cases of organic deafness.

Sedatives may also limit the effectiveness of the EDR test. Depressing effects on electrodermal responsiveness by "suavitil"[27], "gardenal"[32], and barbiturates in general[24] have been reported.

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HEADACHE—A PROBLEM OF MODERN OTOLARYNGOLOGY.[*][†]

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INTRODUCTION.

Because some of the pathways that we in otolaryngology have been travelling seem to have reached blind endings we have partially and inadvertently accepted the idea that otolaryngology as a specialty is dying Although many old ideas and many old practices are dead, a time has arrived not for burial but for re-evaluation — the beginning of a Renaissance.

The otologist if he is to survive, henceforth must be an otophysiologist, a neurotologist and a better otologic surgeon than he ever was to meet today's demands. The laryngologist must be a wide-field neck surgeon, a competent enough neurologist to know that the larynx may be painful without local pathology, and an astute student of the causes of speech and voice defects which have their origins far higher than the hyoid bone or the nasal sinuses. The rhinologist has ceased to operate for sinus infections that never existed. He now understands where the effective realm of nasal surgery lies and what its proper relation is to the nose as a physiologic organ of special sense and respiratory function. He is re-evaluating the relation of gross anatomy and pathology to micro-anatomy, physiology and neurology.

It is with such an altered viewpoint that we must approach the problem of headache.

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THE BASIC MECHANISMS.

When a patient complains of headache he may be speaking of any pain in or about the head or neck. Behind this symptom complex there are, grossly, two mechanisms. The purely sensory mechanism involves only the pain sensory tracts. These convey pain sensation from their source in the periphery directly to the sensorium, or by reference they may project sensations of pain to terminals at a related but remote point. Thus an inflamed lower tooth may convey its message via the mandibular division of the Vth nerve directly to the sensorium to be identified as an aching tooth; or it may by reference to the auriculotemporal branch of the mandibular nerve be projected to its terminals to be identified not as a toothache but as an earache. The pain of an inflamed maxillary sinus supplied by the maxillary division of the Vth nerve may be referred to the area ordinarily associated with the frontal sinus which is of course supplied by the first division of the Vth nerve.

The second or vascular mechanism consists of the pain sensory tracts originating in the blood vessels of the head and neck. These also may convey pain sensations directly to the sensorium, or by reference they may project them via the peripheral sensory system to a remote point; thus edema of the wall of the carotid artery may cause severe pain at the site of the edema. The enlarged and painful vessel may be seen and palpated, and it may be mistaken for an enlarged painful lymph gland; or the pain from this vessel may be referred to the shoulder and arm of the same side.

In explaining the vascular mechanism of headache, Wolff[1] postulates by experiment that vascular headaches are caused by distention of the extracranial vessels, distortion of the venous sinuses, or distortion, displacement, or dilation of the intracranial arteries.

By recording and comparing the immediate effects of intravenous injection of histamine on the cranial and extracranial arteries he concludes that histamine headache immediately following the use of histamine is the result of stretching of the intracranial vessels. The explanation, however, does not seem to apply to the delayed clinical histaminic cephalalgia described by Horton.

Hilger[2] has clarified the explanation of the mechanism of vascular pain by showing that spasm of the vasa vasorum of an artery produces intrathecal edema of that vessel. This edema stretches its coats and so produces pain as surely as does the actual vasodilation postulated by Wolff. Thus the actual basic mechanism is vaso-spasm and not vaso-dilation. An understanding of its *modus operandi* via the sympathetics is grasped readily as the sympathetics are the vasoconstrictors.

CLINICAL DISCUSSION.

We must recognize five clinical conditions that will produce otorhinologic headache.

I. Sinusitis:

Nearly every case of headache coming to us, comes self-diagnosed as sinus headache or simply as "sinus condition," a term as vague to me as to the patient who uses it. Although the present swing of the pendulum leads us away from too frequent diagnosis of sinusitis we must not lose sight of the fact of its existence. While it is not the most frequent cause of otorhinologic headache, it does occur with fair frequency; the headache that it produces may be very severe and may be the indication of serious illness.

There is a significant diagnostic difference between the location of the pain and the location of the tenderness in headache of sinus origin. The location of the pain may be very deceptive. For example in our own experience frontal headache is caused by maxillary sinusitis far more frequently than by frontal sinusitis. The location of tenderness, however, is of marked diagnostic value; thus tenderness at Ewing's point on the floor of the sinus at the inner upper angle of the brow almost invariably means frontal sinusitis.

The cause of the pain may be a blocked sinus ostium with increased positive or negative pressure within the sinus. Based on theoretic considerations it has been said that vacuum sinus headache is impossible of existence. One has only to insert
a needle into a maxillary sinus tha
and literally to see the pain vanis
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McAuliffe, Goodell and Wolf[3] have shown the site of sinus pain to be in the inflamed and edematous duct or ostium and in the inflamed and swollen areas immediately surrounding the sinus opening on both its intrasinal and its intranasal sides. While the *cure* of sinus pain obviously rests on ablation of the underlying infection and so is outside the purview of this discussion, much *relief* may be secured by shrinkage *and anesthesia* of the area in and about the ostium or duct—the source of the pain impulses.

II. Neuritis:

It is in the field of otorhinologic neuritis that most failures to find the cause and to render relief for otorhinologic headache occur.

In general there are three varieties of neuritis of otorhinologic concern:

The first of these is the result of pressure contacts within the nose. These contacts occur between the middle turbinate and the septum or the lateral nasal wall. The headache of this origin varies in distribution with the location of the contact. In general it may be said that the farther forward the contact the farther forward the pain; the farther posterior the contact, the farther posterior the pain.

The second variety of neuritic headache is the "syndrome of the nasal (spheno-palatine) ganglion neurosis" first described by Sluder in 1908 and 1910.[4,5,6] This is a very real and a frequently present clinical entity, and failure to recognize it constitutes a grave clinical error.

The symptoms of this disturbance are widespread, and we will presently understand the reason for this. Pain may radiate to the head, usually keeping below the line of the hat band, and felt most especially in the lower temporal area, the ear, mastoid and occiput. It frequently involves the nape of the neck, the side of the neck and even the shoulder and arm. It may extend to the trunk or leg.

There may be accompanying lacrimation with itching or pain of the pharynx or palate. True cricoidynia, tightness of the throat, especially on swallowing, and even dysphagia are quite common.

In the presence of this disturbance there is almost always tenderness at Sluder's point on the mastoid posterior to the external auditory meatus. I once stood helplessly by as a general surgeon opened a normal mastoid to relieve this symptom which was screaming its own diagnosis.

Sluder relieved these symptoms by intranasal topical or injection anesthesia of the spheno-palatine ganglion area. On the basis of the neurological concept of the time that there were synapses of both the sensory seventh fibers and the sympathetic fibers from the carotid plexus in this ganglion, Sluder attempted to explain the entire process of the symptoms and their relief. As the prime cause of the neuritis he postulated infection, active or latent, in the sphenoid sinus or the posterior ethmoid cells, because of the close anatomical relation of the Vidian nerve to these structures.[7]

As soon as it was shown that all these synapses were not present in the ganglion and that spheno-ethmoidal infection was not always present, Sluder's entire work (both his observed facts and his supporting theories) was attacked and, unfortunately, discredited. This is quite understandable as all of us feel insecure in the presence of a clinical observation that does not seem to rest on accepted anatomic and physiologic bases.

Today with a better understanding of the mechanism of the autonomic nervous system we are on firmer ground, and Sluder's work must be re-evaluated.

On the central side of the ganglion are the sympathetic, the parasympathetic and the sensory supply (see Fig. 1.).

The sympathetic supply consists of non-synapsing postganglionic adrenergic (vasoconstrictor), cholinergic and (most probably) sensory fibers from the cervical sympathetic ganglia via the carotid plexus and the deep petrosal nerve and the Vidian nerve to and through the sphenopalatine ganglion.

The parasympathetics consist of secretory and vasodilator fibers which arise in the nucleus of the VIIth nerve and which reach the ganglion via the great superficial petrosal nerve.

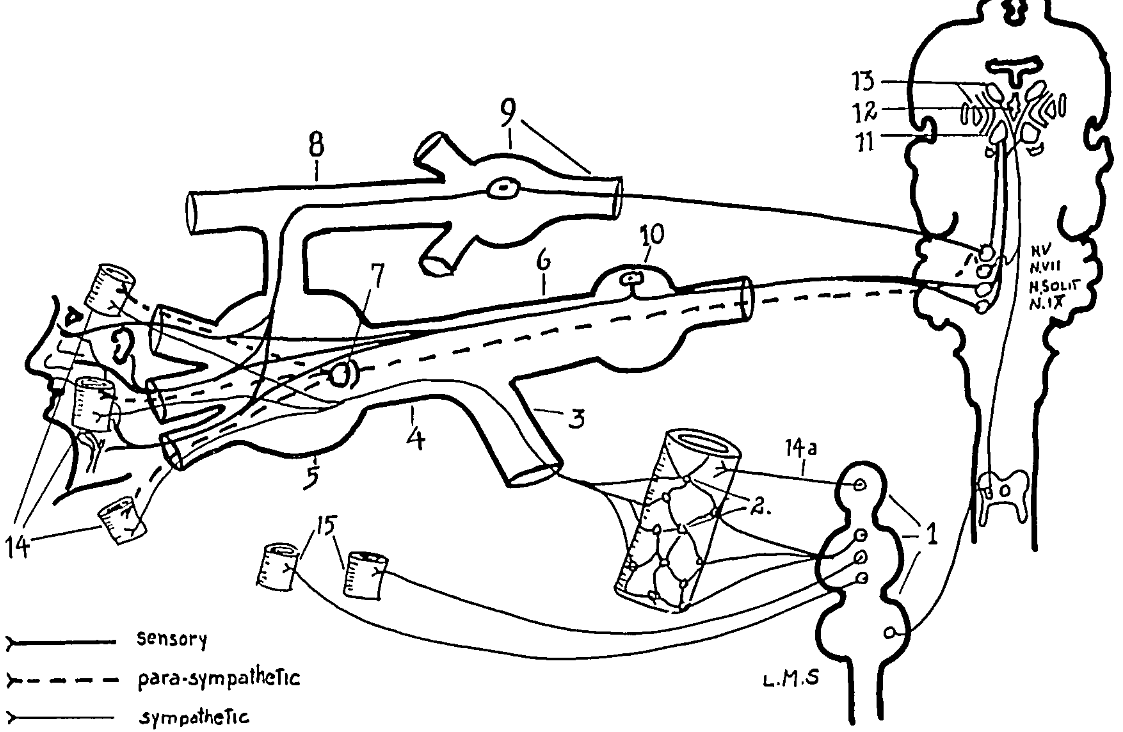

FIG. 1: A DIAGRAMMATIC REPRESENTATION OF THE AUTONOMIC PAIN TRACTS. (The structures are numbered in the order of their mention in the text.) 1. The Cervical Sympathetic Ganglia; 2. The Carotid Plexus; 3. The Deep Petrosal Nerve; 4. The Vidian Nerve; 5. The Sphenopalatine Ganglion; 6. The Great Superficial Petrosal Nerve; 7. A Parasympathetic Synapse Within the Sphenopalatine Ganglion; 8. The Maxillary Division of the Vth Nerve; 9. Semilunar Ganglion and Trunk of the Vth Nerve; 10. The Geniculate Ganglion; 11. The Thalamus; 12. The Hypothalamus; 13. The Basal Ganglia; 14. The Vessels of the Head, Face and Neck (Including The Carotid); 14a. Auxiliary Fibers Direct to the Carotid; 15. Vessels of the Limbs and Trunk.

These fibers apparently are the only ones having synapses within the ganglion.

The sensory fibers coming from the periphery travel centrally through the ganglion without synapsing there and then, either via the maxillary division of the Vth nerve to the semilunar ganglion, or via the great superficial petrosal nerve and the geniculate ganglion (the so-called sensory VIIth nerve), to the nucleus solitarius and the nucleus of the IXth nerve.

The peripheral origins and distribution of these nerves leading to and from the ganglion on its peripheral side include the orbit, the ethmoid and sphenoid sinuses, the nasal cavity (both septum and turbinates), the nasopharynx, the hard and soft palates, the tonsil and finally the larynx (via the IXth nerve fibers of the sensory VIIth).

We now find that we are in a position to understand much that Sluder could not understand.

There are untold numbers of reflex arc fibers, either uninterrupted or synapsing in the ganglion, which pass through that structure. It is a great crossroad of major trunk lines as well as a lesser relay station. Reflex impulses pass centrally over the fibers of the fifth and the sensory seventh nerves to the thalamus and the hypothalamus, and efferently from the cerebral cortex via the pyramidal system and from the lower motor centers of the basal ganglia to the motor seventh nucleus and to the thoraco-lumbar sympathetic system—most especially to the superior, the middle, the stellate and the lower cervical ganglia. From these latter ganglia impulses travel via the sympathetic fibers of the carotid plexus, through the sphenopalatine ganglion to the vasa vasorum of the arteries of the face, head and neck, or directly from the cervical ganglia to limb and trunk arteries. It is these impulses, vasoconstrictor in type, which produce Hilger's type of vascular pain —the pain perceived in this syndrome.

A frequent accompaniment of this headache is myalgia of the sternomastoid or the trapezius muscle which is produced by a localized round cell infiltration, probably surrounding a vessel whose vasa vasorum are in spasm. In these cases the pain is referred to the insertion of the involved muscle—that is to either the mastoid process or to the occiput, whereas the tenderness is in the belly of the muscle involved at the site of the infiltration. When the sternomastoid is involved the localized tender infiltration must not be mistaken for the true carotid pain of Hilger. Relief is afforded by heat and gentle massage along and in the direction of the muscle fibers.

Sluder's syndrome may be accompanied by vasomotor labyrinthitis. This will be discussed later with psychogenic or tensive headache. Here in contrast to psychogenic headache and vertigo there is a local anatomic reason for the pain as well as the vertigo.

The physical causes for sphenopalatine ganglion neuritis are a contact between a far posterior septal spur with the posterior tip of the middle turbinate; inflammation of the posterior group of sinuses, and very frequently, because of a marked concavity of the septum or an atrophy or absence of the posterior tip of the middle turbinate, the sphenopalatine ganglion area is exposed to an inspired blast of irritating raw air.

The diagnosis of any of these headaches of neuritic origin lies in the repeated relief of the pain by anesthesia of the points of contact or the terminal filaments of the nerves involved or the ganglion area.

The treatment is indicated immediately by the diagnostic procedure. This treatment consists of interruption of the afferent impulses from the contact pressure or from the irritated ganglion. Repeated anesthesia of the contact or ganglion area may in itself be effective without any further procedure. Infraction or exfraction of the offending turbinate or its electrocoagulation may be required. Submucous resection of the nasal septum is a frequent requirement. The resection should be complete, not only to remove the contact or to correct a faulty breathway, but also to act as a decompression procedure, so that when the turbinates later engorge physiologically they will not impact against a rigid and unyielding wall.

Frequently after surgery has been performed sessions of anesthesia may have to be continued for a short time if the pain is not relieved completely by the operation. Here a "habit pain pattern" has been set up in the reflex arc that persists after the removal of the physical irritant. It is the interruption of this arc by anesthesia that finally effects the cure. When topical anesthesia fails to relieve the pain, blocking of the stellate ganglion area by the method of Alexander[8] should be done. This is highly effective as the blocking agent in this particular procedure reaches the lowest sympathetic fibers, although the accompanying Horner's syndrome may be disturbing to an introspective patient.

The third and last neuritic type of headache seemingly has not been described previously although it has been hinted at under such terms as "atypical neuralgia" or "atypical face pain." I have not worked out definitely the tracts involved, but I feel that this headache is based on the conception of White, Smithwick and Simeone that there are pain tracts in the sympathetics.[9] For this reason I have spoken of it as sympathetic neuritis. Eventually it may be demonstrated to be only a variation of vascular pain.

It is characterized clinically by a peculiarly indescribable nagging pain, usually starting in or about a tooth that evinces no apparent pathology. The tooth usually is extracted against the better judgment of the dentist, because of the insistence of the patient who is terribly annoyed by the pain. If the dentist holds out against the extraction, and the patient endures the pain which no treatment seems to relieve, the pain usually disappears; but if the tooth is extracted, mischief is afoot: the pain increases; then follows extraction of one of the abutting teeth or perhaps of both of them. This is followed in turn by repeated curetment of the socket and biting away of the alveolar ridge.

Blessed is the patient if this occurs in the lower jaw because no sinus abuts it. If, unfortunately, it is an upper tooth the antrum is next attacked; then the ethmoids, and finally the sphenoid. With each succeeding surgical procedure the pain becomes distressingly worse. I know this, because I performed the last three of a series of operations suffered by the patient in the first case that I ever saw and recognized.

I reoperated a foully infected antral pocket after the victim had had all molars and premolars on the affected side extracted and then had suffered a combined radical antrum and ethmoid exenteration. Later I performed a sub-mucous resection and finally evulsed the infra-orbital nerve which resulted in complete second division anesthesia in the affected area but without relief of the pain. It was after the last procedure that it finally dawned on me that the pain was following the blood vessels and not the second division of the Vth nerve.

With this recognition of a clinical entity other cases soon appeared in the practice. It is not a frequently met situation, but if it can be recognized, preferably before the first dental or surgical attack, the patient and the doctor will be happier for it. It does seem to be self-limited if only the patient can be prevailed upon to leave it alone; otherwise I know of no treatment for it.

III. Psychogenic or Tensive Headache:

This headache is the twin sister of psychogenic vasomotor labyrinthitis which produces vertigo and hearing disturbances, and they frequently appear together.[10] It operates via the same mechanism as sphenopalatine ganglion neuritis, following the same anatomic vicious circle, but its point of origin lies not in the ganglion or in the periphery but in the diencephalic structures—notably the thalamus and the hypothalamus. Here the cure rests on two factors. If possible the psychic cause should be discovered and made clear to the patient *by the otolaryngologist* if at all possible, at whatever cost of time and effort to him; the patient's cooperation should be obtained in rearranging his work, worry and eager-beavering, and in altering his responses to his problems of life. Pharmacologic interruption of the vicious circle by anesthesia of the ganglion area is the second and equally important factor in effecting the cure. Again it is the mechanical or pharmacological breaking of a "pain habit pattern" that may be necessary for a cure. Either factor without the other may prove to be of little avail.

IV. Histaminic Cephalalgia:

This headache first described by Horton[11] is a definite clinical entity which must be recognized by the otolaryngologist in his consideration of headache, as it may well be mistaken for neuritic headache. To enter into a discussion of a subject covered so well by Horton in his various publications is unnecessary. Suffice it to say that this headache is met frequently, and its symptoms are so severe as to be disabling. When correctly diagnosed and treated, the results are so dramatically satisfactory that it sires a patient whose gratitude knows no bounds.

V. Neoplasms:

These offer such a wide range of pain symptoms that a monograph would be required to describe them; but three facts must be mentioned:

Tumors of the fossa of Rosenmueller—usually epidermoid carcinoma, transitional cell type—may produce typical Sluder's syndrome. Let us always examine this region for such a possibility in the presence of the syndrome of sphenopalatine ganglion neuritis.

Tumors of the larynx may have as their first symptom severe otalgia, and unexplained ear pain calls for meticulous laryngoscopy.

Finally, cases of malignancy of the nasopharynx that have been treated with radiation therapy often develop severe unilateral headache after most of the reaction has subsided. For some as yet unexplained reason anesthesia of the ganglion area seems to be the most effective means of relief.

CONCLUSION.

We find that another expanding field has been reopened to otolaryngology. The physician who can relieve what may well be the most widespread and most frequent complaint of modern man is in an enviable postion. Our strategic position in the reawakened field of otolaryngology where we are apt to be the first physician to see the patient with headache and where we are the best equipped of all branches of medicine to understand and to conquer the problem, makes us doubly to be envied.

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TREATMENT OF ACUTE SINUSITIS WITH PENICILLIN AND THE INTRAMUSCULAR USE OF STREPTOKINASE.[*]

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Acute sinusitis is a diffuse inflammation of the mucosa of the sinuses with hyperemia, edema and serous exudation. The edema produces occlusion of the ostium of the sinus, the normal route for escape of secretions. A favorable therapeutic response will be obtained in many patients treated early in the course of the disease with a combination of chemotherapeutic agents, antibiotics, local application of vasoconstrictor drugs and sedation for relief of pain. Progression will be seen in some patients in spite of such measures. The ostium will remain blocked, the edema will increase in amount, and pus will collect. If drainage is not provided, subperiosteal abscess, osteomyelitis, meningitis, extradural abscess, intracerebral abscess or chronic sinusitis may occur. The patient who fails to respond to conservative measures must be treated by surgical drainage of the sinus to prevent complications.

Recently it has been shown that the intramuscular injection of streptokinase will reverse the inflammatory state and reduce edema.[1] The morphologic changes of inflammation seen

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[*] From the Surgical Service, Veterans Administration Hospital, Fort Howard, Maryland (Dr Miller, Dr Surmonte and Dr Ginsberg) and the Lederle Laboratories Division, American Cyanamid Company, Pearl River, New York (Mr Ablondi).

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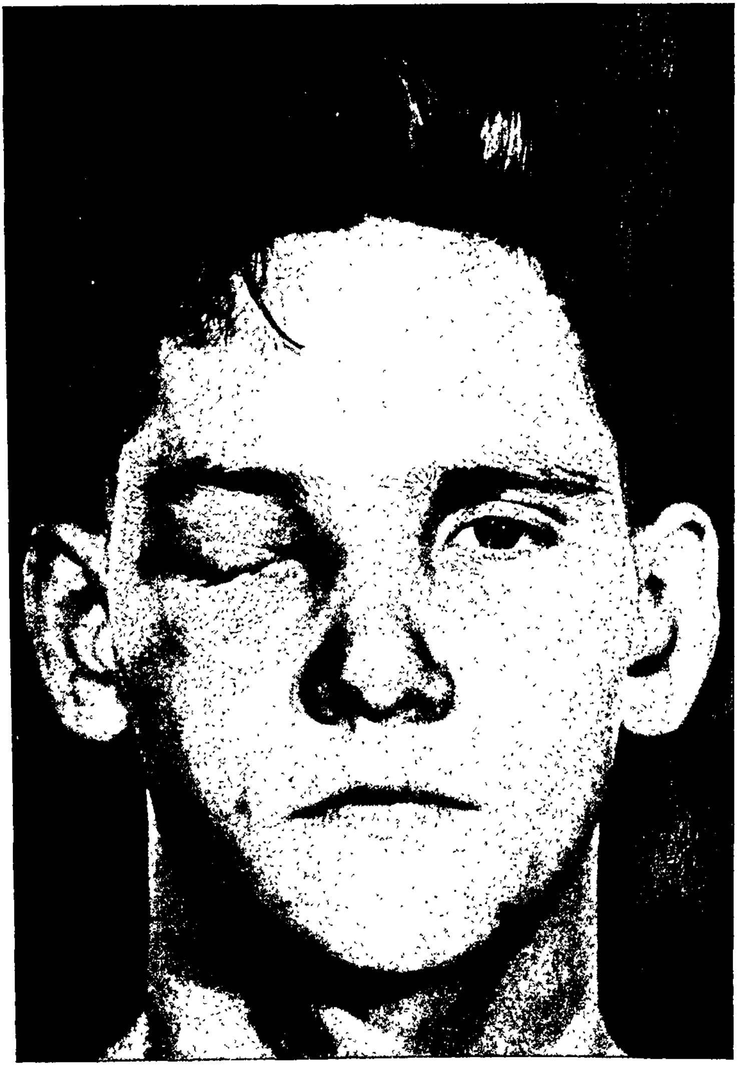

Fig. 1. Appearance of the face on September 17, 1954.

in the tissues are the mechanism by which an injurious agent is localized in the body. These barriers not only prevent spread of infection, but also stop the adequate delivery of anti-bacterial substances from the rest of the body to the infected area. Streptokinase will remove some of these hindrances so that greater amounts of anti-microbial drugs can

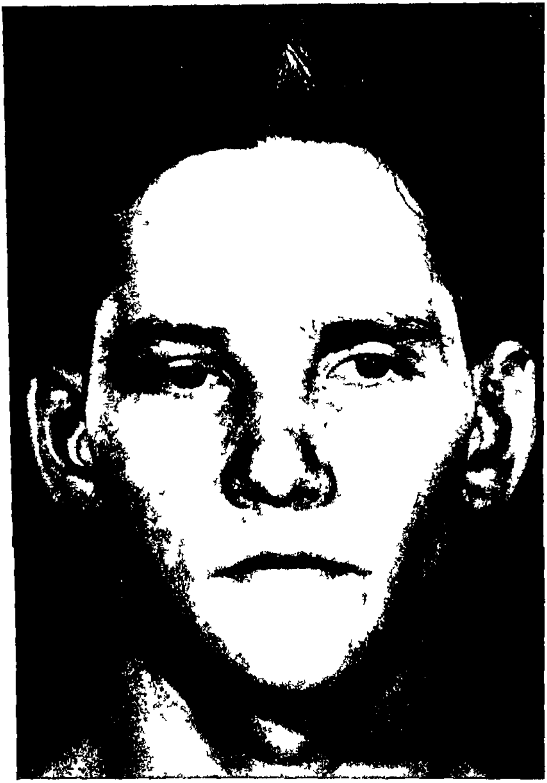

Fig 2 Appearance of the face on September 20 1954

go to the involved site The intramuscular injection of streptokinase cannot be used without the concurrent administration of anti-bacterial agents, or spread of the infection might be facilitated

Varidase, composed of streptokinase and streptodornase, was dissolved in physiologic saline so that the final concentration of streptokinase was 10,000 units per cc. The solution was stored in the refrigerator when not being used. A fresh solution was made daily. The effects achieved by the injection of varidase were ascribed to streptokinase, since the observed therapeutic effects were not produced by the intramuscular administration of a purified preparation of streptodornase.

Case Report—A 20-year-old white man was admitted to the hospital on Sept. 16, 1954, with an acute right frontal sinusitis of five days' duration, associated with periorbital edema and occlusion of the palpebral fissure. Severe headaches, chills and fever had been present. The patient had received three injections of an unknown amount of penicillin by his family physician without effect.

The temperature was 101.4°F. There was redness and edema of the right periorbital area (see Fig. 1), and tenderness over the frontal sinus. The mucous membrane over the right middle turbinate was red and swollen. Roentgenograms showed clouding of the right frontal and ethmoid sinuses.

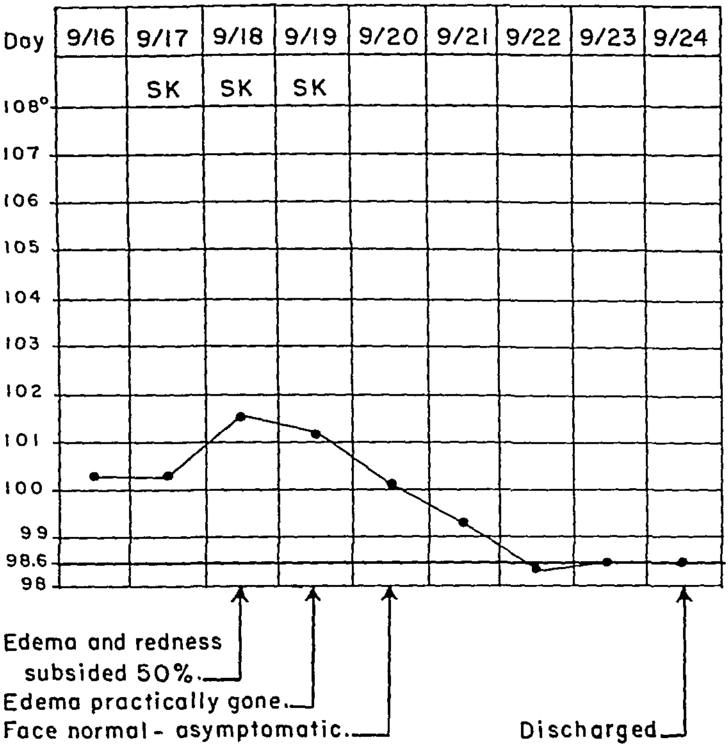

Fig. 3. Clinical course of the patient.

Packing, saturated with [illegible] of 10 per cent cocaine hydrochloride nd 1:1000 epinephrine was put into the right side of the nose, four imes daily, and 20 drops of 0.2 per cent solution of neosynephrine were laced into the same side every three hours from Sept. 16 through Sept. 0. The patient was given [illegible]0,000 units of procaine penicillin G in queous solution and 0.5 gm streptomycin sulfate, both intramuscularly, wice daily from Sept. 1[illegible] through Sept. 24. Intramuscular injections of ,000 units of streptokinase in 0.5 cc of physiologic saline were given wice a day from Sept. 1[illegible] through Sept. 19. On Sept. 18, the periorbital dema had started to subside (see Fig. 2), and the headache had disppeared. By Sept. 19 the patient was asymptomatic, and the edema was ractically gone (see Fig. [illegible]). On Sept. 20 all evidence of infection had isappeared. The patient was afebrile on Sept. 22. The patient was ischarged from the hospital on Sept. 25.

SUMMARY.

Streptokinase administered intramuscularly with the antibacterial drugs was a valuable adjunct in the treatment of a patient with an acute right frontal sinusitis. It is suggested that, because of the beneficial effects observed in this patient, this method can be used in the office management of patients, whose disease is not quite so severe, to prevent hospitalization. Further investigation of this treatment is indicated.

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THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY CONVENTION CRUISE

The Virginia Society of Ophthalmology and Otolaryngology is sponsoring a convention cruise to Havana and Nassau on May 26 to June 2, 1956. Sailing from and returning to Norfolk, Virginia, the "*Queen of Bermuda*" will act as the hotel for the trip. Fare for seven days, $165.00 and up per person. Make reservations with United States Travel Agency, Inc., Washington, D. C.

CONGENITAL ATRESIA OF THE EXTERNAL AUDITORY MEATUS.*

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Surgical correction of congenital atresia of the external auditory canal has been attempted many times over the years with very little success before 1947, when Pattee[1] reported on five cases and showed good results in four of them. Prior to that time Fraser[2] and Richards[3] had reviewed the embryological and anatomical anomalies that are generally found.

The inner, middle and external ears develop from different anlages and, therefore, congenital malformations of the canal are rarely associated with inner ear changes. The inner ear develops first from an invagination of the ectoderm that forms the otocyst from which the cochlea and labyrinth develop.

The external ear and Eustachian tube develop from the first pharyngeal pouch. Simultaneously, there is an outpocketing of the entoderm to form the Eustachian tube and a pitting of the ectoderm at the site of the future external auditory meatus. A cord of cells is formed from the ectoderm to the entoderm, where a plate forms just lateral to the middle ear cavity. This cord of cells later canalizes from within out, leaving the tympanic membrane at the site of the junction of the ectoderm and entoderm. The auricle forms from six mounds developed from the first and second branchial arches around the first branchial groove. The malleus and incus develop from Meckel's cartilage of the first branchial arch, and the stapes arises from Reichert's cartilage of the second branchial arch.

The os tympanum develops directly from membranous bone as a U-shaped bone surrounding the ectodermal core previously described, and it subsequently forms the sides and floors of the bony external auditory canal.

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* Read before the New York Academy of Medicine, Section of Otolaryngology, May 18, 1955.

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Failure of the ectodermal core to canalize results in a deformed tympanic bone, and an overgrowth of bone occurs to fill most of the space normally occupied by the external auditory meatus.

Associated with congenital atresia of the canal are varying degrees of deformity of the auricle and the incus and malleus. There are also varying degrees of formation of the tympanic cavity, and an associated pneumatization of the mastoid bone.

The surgery of choice depends upon the extent of the malformation within the tympanic cavity. Pattee believed that previous failures to correct the hearing were due to neglecting the fact that the incus and malleus were usually deformed and that they were probably fused into position, thereby immobilizing the stapes; therefore, he removed the incus and checked the motion of the stapes in each case before applying a Thiersch graft to form a pseudo-tympanic cavity.

In 1947, also, Ombredanne[4] described two cases in which he did a similar operation and a fenestration in one stage with excellent results in one case. He later reported[5], in 1952, on 33 cases done with the same one-stage technique.

Meanwhile, in this country, several authors including Altmann[6] suggested the possibility of doing a fenestration in one or two stages for the same deformity. Woodman[7] in 1952 reported a case in which he first operated using the Pattee method but carefully skeletonized the horizontal semicircular canal. At a later date, following little or no improvement in the hearing, it was a relatively simple procedure to raise a flap and do a fenestration with excellent functional result.

On the service of Dr. Ward C. Denison at the New York Eye and Ear Infirmary, we recently operated on a seven-year-old boy with a complete atresia of one ear and an almost complete occlusion of the other. We are presenting his case, further to emphasize the fact that in congenital anomalies any combination of malformations can occur, and each case must be individualized.

Case Report: (H. H.) This seven-year-old white male was sent home from school with a note stating that the teacher believed that his difficulties in school were caused by his inability to hear properly. He had no complaints of pain at any time, and only by careful questioning were we able to elicit the fact that he occasionally had drainage from the left ear. His past medical history revealed no previous illnesses. His tonsils and adenoids had been removed at the age of four.

Physical examination revealed a well nourished, well developed boy with abnormalities limited only to the ears. Both auricles were perfectly formed and properly placed. The right ear showed a shallow depression leading to a blind pouch in place of a normal external auditory meatus. In the left ear there was extreme narrowing of the meatus to approximately 1 mm. in diameter, allowing no view of the tympanic membrane.

Radiographs of the temporal bones revealed no discernible evidence of the external auditory meatus with partial pneumatization of each mastoid.

Audiogram showed an average 45 db loss in the speech range of the right ear, and a 35 db loss in the speech range of the left ear. Because the right ear had the poorer hearing, surgical intervention of that side was proposed.

On June 16, 1954, under general anesthesia, a right endaural incision was made, and the mastoid bone was exposed. The cortex overlying the antrum was removed. The periantral cells were exenterated and the horizontal semicircular canal was skeletonized. Using the canal as a landmark, the zygomatic arch, posterior and superior canal walls and the bone filling the area of a normal external auditory meatus were removed with the drill and cutting burrs.

The boy was found to have a complete soft tissue atresia, and the skin was excised anteriorly to what seemed the normal anterior margin of an external auditory meatus. The bony occlusion was of an hour-glass shape, and did not continue down to the tympanic membrane. The membrane itself was in an infantile (or more horizontal) position, and was not of a normal color or consistency. It contained a large perforation. The ossicles appeared to be normal and were left in place. The entire cavity was then lined with a split thickness graft taken from the thigh, and was packed with cotton pledgets.

The right ear healed quickly, and in less than two months there was no further discharge, and the hearing had improved to an average of 20 db below normal, an increase of 25 db.

On Sept. 15, 1954, the left ear was operated on in a similar manner. On the left side there was not a complete occlusion of the canal, but rather only a marked stenosis. Cholesteatoma was found in the antrum and attic with a large tympanic membrane perforation. All mastoid air cells were carefully exenterated, and the ossicles were cleaned but left in place. Again a split thickness graft was applied.

Recovery on the left side was slower than that of the right, because of cholesteatoma and infection; however, in four months the entire cavity was healed with dry perforations of the tympanic membrane.

The last audiogram, taken on Feb. 12, 1955, showed an average loss of 15 db in the speech frequencies of each ear. This amounts to a gain of 30 db in the right ear and 20 db in the left ear. This boy has improved in school to such an extent that he has been advanced to the stage of his normal

DACRYOCYSTORHINOSTOMY: SIMPLIFIED PROCEDURE.*

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Numerous operative procedures have been advanced over the years for the relief of chronic dacryocystitis, mucocele and epiphora; in fact, it would be difficult to offer any operation which at least has not been described as a part of some often rather extensive or complicated procedure. The many operations described only emphasize the lack of a generally accepted procedure.

From the many operations offered and from a varied personal experience over the past thirty-five years, in which I have had a particular interest in this work, I am convinced that the operation can be done simply, without suture of the lacrimal sac to the nasal mucous membrane or without transplanting any part of the sac into the nose or extending any material such as tubes, sutures, or packing into the nasal passage.

INDICATIONS.

Operation is indicated in any chronic dacryocystitis or chronic epiphora where the inferior canaliculus is open as is indicated by a return of tear or pus into the eye when pressure is made over the lacrimal sac. The obstruction is usually at the mouth of the lacrimal duct where it often narrows as it leaves the lower portion of the sac. Age: my patients have ranged from 4 to 78 years

SURGICAL ANATOMY.

The lacrimal sac (see Fig. 1) is on the average 12 mm. long[1] and 2 to 5 mm. wide, and the dome is usually from 2 to 5 mm. above the exit of the orifice of the canaliculi; thus, it is evident that the maximum opening of only 1 cm. can be

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* Read at the meeting of the Middle Section American Laryngological, Rhinological and Otological Society, in Cincinnati Ohio January 16, 1956

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made in the vertical direction of the medial surface of the sac at operation. With a maximum width of only 2 to 5 mm. it is true that any opening much wider than 7 mm. in the bone of the lacrimal fossa into the nose is of little importance.

Usually only very thin lacrimal bone (see Fig. 2) (often of paper thickness) and the posterior edge of the frontal process of the maxilla bone form the medial bony wall of the lacrimal fossa (approximately half and half). Very commonly, however, according to Schaeffer[1] two or more anterior ethmoid cells (frontal and infundibular) immediately adjoin the dorsal and medial aspect of the lacrimal sac; even a dehiscence may be present. He states that "even more common is the extension of anterior ethmoid cells over the medial or nasal side of the lacrimal sac and the beginning of the nasolacrimal duct." The rhinological problem thus becomes more important in dacryocystorhinostomy than the ophthalmological problem of dealing with the lacrimal sac. The entire wall of the lacrimal fossa may be pneumatized by ethmoid cells which must be eliminated at operation.

Another rhinological problem of importance is that of a deviation of the nasal septum which must be corrected at the time or preceding the dacryocystorhinostomy.

The anterior lacrimal crest is of the maxillary bone while the posterior crest is of the lacrimal bone.

OPERATION.

Local anesthesia: Premedication in adults consists of Nembutal or Seconal, grains one and a half, given orally, and morphine, grains 1/6, plus scopolamine, grains 1/150, intramuscularly, forty-five minutes to one hour before operation.

One per cent novocaine containing adrenalin (1:1000) fifteen drops to the ounce is injected along the medial wall of the orbit (1 cm. above the inner canthus) toward the anterior ethmoid nerve. A second injection extends toward the infraorbital foramen and along the line of incision.

Cocain powder and adrenalin is applied to the mucous membrane of the middle meatus.

Incision: A straight line incision is made midway between the midline of the nasal bridge and the inner canthus, as any incision made nearer to the canthus may produce an irregularly drawn scar. The incision extends 1 cm. above the canthus and 2 cm. below.

Frequently the inexperienced operator carries the incision too high, even into or below the eyebrow, because he loses sight of the fact that the sac lies almost entirely below the canthus. So high an incision results in unnecessary bleeding and an awkward approach to the sac.

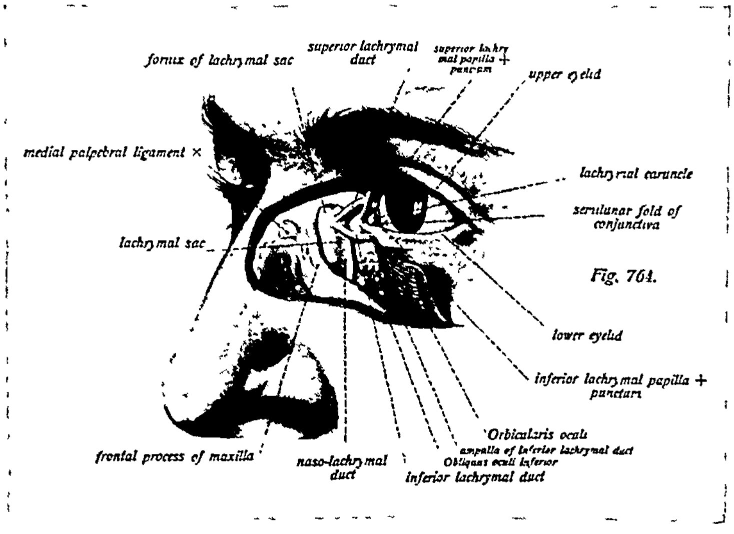

Fig. 1. Lateral wall of the lacrimal sac. (From Atlas and Text Book of Human Anatomy, p. 269, by Sobotta McMurrich. Saunders and Co.)

The incision is carried immediately through the soft tissues and periosteum to the bone.

The soft tissues and periosteum are elevated, together with the medial palpebral ligament which is not cut. The elevation is carried medially by means of a fairly sharp submucous elevator, starting first over the frontal bone above the lacrimal fossa exposing the upper end of the anterior lacrimal crest and the upper portion of the fossa. The posterior crest is exposed, but the sac is not elevated beyond this point. As the elevator is moved downward into the fossa the nasal wall of the sac is easily exposed well into the mouth of the lacrimal duct.

The attachment of the lateral wall (see Fig. 1) is not disturbed, as this attachment will serve to keep the sac in a better position post-operatively

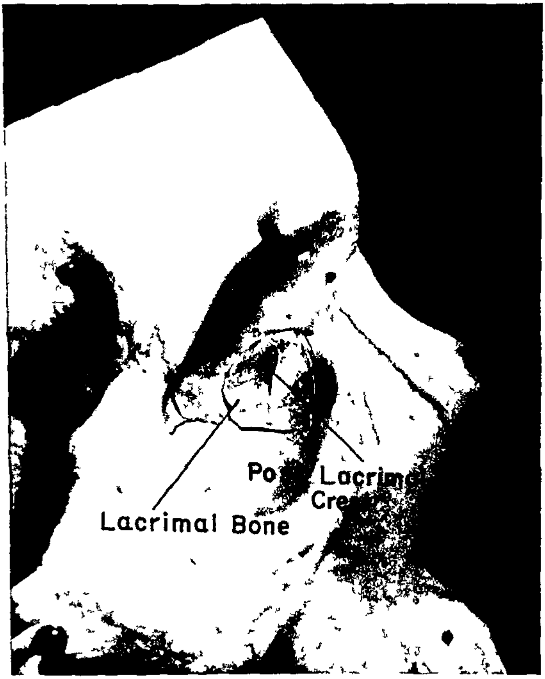

Fig 2 Lacrimal bone showing its posterior lacrimal crest

Bone fenestration. The lacrimal bone (see Fig. 2) is often of paper thickness and can easily be opened by means of a sharp 2 or 3 mm. mastoid curette (Spratt) held firmly in one hand while being turned medially and then outwardly with the opposite hand. All the bone necessary may be removed by means of the curette without opening the nasal mucous membrane. The bony opening is extended well into the mouth of the lacrimal duct and this extension is important. This opening is also extended to the posterior lacrimal crest and including the crest, especially in its lower portion. The bone is removed upward to the level of the dome of the sac.

The medial edge of the anterior lacrimal crest may be shaved away for 1 to 2 mm. well down into the mouth of the duct. I do not attempt to remove heavy thick bone of the maxillary process which adds only to the formation of granulations in healing.

The mucous membrane of the nose, if not already opened, is incised and cut away cleanly just to the size of the opening previously made in the bone; thus, the nasal mucosa over the lacrimal sac is sacrificed completely. A Hartman middle ear, through-and-through biting forceps is very helpful in the final trimming of the soft tissues, as the edge must be cut clean and not dissected up beyond the edge of the bony opening. If anterior ethmoid cells intervene, a second bony wall is dealt with.

The medial wall of the lacrimal sac is lifted by means of a dural hook and opened in its midline with a small knife. The incision is carried in the vertical plane well into the mouth of the lacrimal duct and upward within 2 mm. of the dome of the sac (see Fig. 3). A horizontal incision is then made the full width of the sac at the upper end of the vertical incision, 2 to 3 mm. from the top of the dome, which will expose the mouth of the common duct of the canaliculi.

A second horizontal incision is made at the lower end of the vertical incision as far into the mouth of the duct as possible as it is in this area that the opening into the nose can be established well below the anterior end of the middle turbinate. Here one has to deal only with thin bone and nasal mucosa.

The incisions in the nasal wall of the sac having been carefully and widely procured, the long posterior flap of the sac is laid into the nasal opening. The anterior flap is turned anteriorly and held in position by means of two 4-0 plain catgut sutures carried into adjacent soft tissues on the medial side. I have, however, laid this flap into the nasal opening without suturing, with excellent results; once the sac is opened widely there seems to be no tendency for the sac to reform. It is the nasal opening which offers our chief problem.

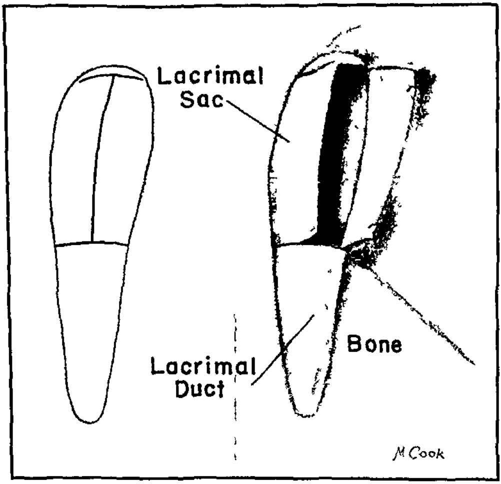

Fig 3 Diagrams of incisions made in the nasal wall of the lacrimal sac

The soft tissues are laid back into position and the skin closed by two figure-eight fine silk sutures which are removed on the fourth day; often cut on the second day.

Vaseline ointment is applied to the eye and a light bandage applied for twenty-four hours.

No packing is placed in the nose, and the patient leaves the hospital on the day after operation.

After Care: The patient is up on the first day. The sutures are removed by the fourth day, sixth in children, and the nose examined for blood clots.

Usually tearing stops immediately, and by the seventh day gentle pressure may be applied intermittently by the patient to milk the sac as a guard against a retarded flow of tears into the nose. A 5 per cent solution of silver nitrate may be applied at the end of a week or ten days in the middle meatus. Should some tearing occur a probe is passed through the inferior canaliculus and the distal end moved upward in the nose to separate any adhesions that might have occurred. Should this procedure fail and the opening into the nose close, the following procedure will usually re-establish the nasal opening. It is a scarring of the nasal tissues that produces the closure—not a reformation or closing of the wall of the lacrimal sac.

The procedure of reopening the tract consists of injecting the area of the sac with 1 per cent novocain and cocainizing the nose. A probe is passed through the inferior canaliculus and pressed firmly against the inner wall of the nose. While an assistant holds the probe firmly in this position a right angle tonsil knife with a short blade is used to cut down on the probe as it presents in the middle meatus. A triangular incision is made with the base of the triangle downward. The probe is passed at five to seven day intervals for several weeks and the distal end moved up and down in the nose to prevent a new membrane from forming. The patient is also instructed to apply intermittent pressure against the sac to produce a pumping or intermittent suction effect.

In most cases no after treatment is required, especially where the nasal mucosa has not been unduly traumatized beyond the opening made in the bone. None of my cases have been complicated by hemorrhage, and often no treatment whatever has been applied to the nose following operation.

SUMMARY.

I have depicted in a practical way the surgical anatomy and its importance and limitations in dacryocystorhinostomy.

I have emphasized the wide opening of the lacrimal sac and stressed the importance of its extension well into the mouth of the duct.

I have emphasized the importance of a precise opening into the nose with minimal trauma to the edges of this opening and the complete sacrifice of the nasal mucosa over the area corresponding to the sac.

REFERENCE.

1. Schaeffer, J. P.: The Nose and Olfactory Organ. P. Blakiston's Son and Company, Philadelphia, p. 249, 253, 1919.

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY.

The Scientific Meeting of the American Society of Facial Plastic Surgery will be held Wednesday evening, March 7, 1956, at the Biltmore Hotel, New York City. Program will be as follows:

I. Septal Surgery in Rhinoplasty (Rapid Procedure). Dr. Jesse Fuchs—Member Attending Staff, Cedars of Lebanon Hospital and Mount Sinai Hospital, Los Angeles, California.

II. Otoplasty for Protruding Ears (Movie Presentation of Technique). Dr. David Meyers—Professor and Head of Department of Otolaryngology, Temple University, Philadelphia, Pennsylvania.

Discussion. Dr. I. B. Goldman — Rhinoplastic Surgeon, Mount Sinai Hospital, New York City.

III. Use of Thorazene in Anesthesia and Its Application to Rhinoplastic Surgery. Dr. LeRoy W. Krumperman—Professor of Anesthesiology, Temple University Medical Center.

IV. Jacques Joseph's Last Rhinoplasty—Outcome. Dr. Gustave Fred—Consultant Otolaryngology, Beth Israel Hospital and Massachusetts General Hospital, Boston, Mass.

Make reservations for yourself and guests at your earliest convenience. Subscription $15.00 per person, all inclusive. William Schwartz, M.D., Secretary, 224 Lexington Avenue, Passaic, N. J.

BOOK REVIEW.

Bronchography in Children (La Broncografia nel bambino). By Lucio Parenzan and Alfredo Vago. Supplement XXIII of the Archivio Italiano di Otologia, Rinologia e Laringologia, 1955 (In Italian. Summaries in English, French and German).

This is an authoritative contribution to the knowledge of lower respiratory pathology in children.

A new and safe technique of bronchography under general anesthesia is described with excellent results obtained in over one hundred children. Several examples of normal anatomy and tracheobronchial topography are presented. The authors list the indications for bronchography in children as tracheobronchial pulmonary malformations, chronic bronchopulmonary suppuration and primary tuberculosis.

Bronchography means team work between bronchoscopist, radiologist and anesthetist. It becomes the essential item of a triad of diagnostic procedures in lower respiratory pathology: laminography, bronchography and bronchoscopy. The authors suggest that in children even bronchoscopy should be "guided" by bronchography.

The exposition is clear and simple, and many references are given. The monograph is amply documented by outstanding illustrations with explanatory diagrams making the reading extremely easy and enjoyable.

E. J. F.

WASHINGTON UNIVERSITY, SAINT LOUIS, DEPARTMENT OF OTOLARYNGOLOGY.

Offers a Basic Science Course in Otolaryngology to start Monday, September 17, 1956. Complete information about the course may be obtained by writing to Theo. E. Walsh, M.D., Head of the Department of Otolaryngology, 640 S. Kingshighway, St. Louis 10, Missouri.

ERRATA.

The following legends were inadvertently omitted from the article on Acetylcholinesterase Activity in the Cochlea, by J. A. Churchill, M.D., H. F. Schuknecht, M.D., and R. Doran, in the January, 1956, issue of The Laryngoscope:

FIGURE 1 Photographs taken through the dissecting microscope

Upper· Experiment 2 View of treated (T) and control (C) cochleae placed adjacent to each other showing band of copper sulfide precipitate (arrows) in the three turns of the treated cochlea

Lower Higher power magnification of the treated cochlea showing the band of copper sulfide precipitate (I) near the margin of the osseous spiral lamina (S)

FIGURE 2 Photographs taken through the dissecting microscope

Upper Experiment 1 In the exposed basal turn of the treated cochlea there are bands of copper sulfide precipitate lying in relation to inner hair cells (I) and outer hair cells (O) The osseous spiral lamina is seen at "S"

Lower Experiment 2 130x Teased specimen from basal turn, not counterstained, showing dense band of precipitate (I) related to inner hair cell row and small discrete deposits of precipitates (O) adjacent to the three rows of outer hair cells

FIGURE 3

Upper Experiment 8 Teased preparation from apical turn, incubated in D F P, counterstained with acid fuchsin, showing dense band of precipitate (I) lying in relation to inner hair cell row There is a small amount of precipitate on outer hair cell rows (O) The osseous spiral lamina (S) and spiral ganglion (G) are seen

Lower Experiment 6 Microscopic section of organ of Corti from middle turn showing large deposit of precipitate (D) located above the inner phalangeal cell which was detached from inner hair cell (I) There is a small deposit of precipitate in the habenula perforata (H) and at the base of the first outer hair cell (O) The pillars are seen at "P" and the tectorial membrane at "T" It should be remembered that considerable post mortem degeneration is present because about 90 minutes elapsed between death of the animal and fixation of the tissues, 45 minutes of which they were incubated at 38° C

FIGURE 4 Photograph taken through dissecting microscope

Upper Experiment 2 Copper sulfide precipitate in the three turns of treated cochlea Notice precipitate on nerve fibres (N) in osseous spiral lamina of apical turn

Lower Experiment 2 Teased preparation from apical turn showing precipitate on the nerve fibres (N) in the osseous spiral lamina The organ of Corti and basilar membrane are missing except for a small portion of the inner hair cell row with dense copper sulfide precipitate (I) The small deposits of precipitate (E) along the edge of the osseous spiral lamina appear to be in relation to nerve endings for inner hair cells

FIGURE 5 Experiment 8 Muscle control All techniques, including photographic factors, held constant

Upper Microscopic section showing deposits of copper sulfide precipitate in the muscle end plates

Center Another section from same muscle treated in addition with D F P to show disappearance of some of the diffuse precipitate believed to be due to non specific cholinesterase

Lower Same, except not treated with substrate acetylthiocholine

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

The Sixth International Congress of Otolaryngology will take place in Washington, D. C., from Sunday, May 5, through Friday, May 10, 1957, under the presidency of Arthur W. Proetz, M.D.

The selected subjects for the Plenary (Combined) Sessions to be held Monday, Wednesday and Friday mornings will be:

1. Chronic Suppuration of the Temporal Bone.
2. Collagen Disorders of the Respiratory Tract.
3. Papilloma of the Larynx.

Outstanding internationally recognized authorities will open the discussion of each of these subjects.

Two types of communications are invited: *1.* Contributions to the discussions of the selected subjects, limited to five minutes. *2.* Original papers, limited to 15 minutes. These should be in one of the four official languages: English, French, German, Spanish.

Motion picture films will be shown continuously except during the Plenary Sessions. There will be both scientific and technical exhibits. Those wishing to submit contributions to the program should communicate with the General Secretary.

Announcement of the Congress has been sent to all otolaryngologists whose names and addresses could be obtained. Additional details concerning registration, housing, entertainment, etc., will be sent to those who have indicated to the General Secretary that they wish further information.

The subscription for Members (physicians) is $25.00 U.S.A. This includes the privilege of attendance at all official Congress meetings except the banquet for which an additional charge will be made. Other persons accompanying Members may be registered as Associates at a fee of $10.00 U.S.A.

An interesting program of social functions, visits to points of interest in and around Washington and post-Congress tours is being arranged. The American Express Company is the official travel agent for the Congress. Their offices throughout the world are available for travel arrangements to the Congress and for post-Congress tours.

All communications should be addressed to the General Secretary, Paul H. Holinger, M.D., 700 N. Michigan Ave., Chicago, Ill., U.S.A.

INDIANA UNIVERSITY MEDICAL CENTER.

The Department of Otolaryngology, Indiana University School of Medicine, offers its annual Anatomical and Clinical Course in Otolaryngology March 26th to April 7th, 1956.

Applicants should address The Post-Graduate Office, Indiana University Medical Center, Indianapolis 7, Indiana.

NEW CLINIC OPENED.

A new Clinic for Reconstructive Plastic Surgery of the Face was opened officially on December 8, 1955, in a new wing of the Manhattan Eye, Ear and Throat Hospital, New York City. Dr. John Marquis Converse is the Surgean-Director of the Clinic.

The staff includes a co-ordinated team of specialists in fields associated with plastic surgery who will evaluate and treat all types of facial disfigurements. A teaching clinic will be developed, and fellowships in plastic surgery are to be made available.

ENDAURAL OTOLOGIC SURGERY COURSE.

A special course in Endaural Otologic Surgery will be given by Northwestern University February 28 through March 26, 1956. Course is limited to eight otolaryngologists. Instruction to include lectures, cadaver dissection, etc. For further information write Dr. Geo. E. Shambaugh, Jr., Dept. of Otolaryn., Northwestern Medical School, 303 East Chicago Ave., Chicago (11), Ill.

NATIONAL SOCIETY MEETINGS.

Schedule of Meetings for 1956:

American Board of Otolaryngology, to be held at the Sheraton-Mt. Royal, Montreal, Canada, May 6-11.

American Otological Society, Inc., to be held at the Seigniory Club, Ottawa, Canada, May 11-12.

American Laryngological Association to be held at the Seigniory Club, Ottawa, Canada, May 13-14.

American Broncho-Esophagological Association, to be held at the Sheraton-Mt. Royal, Montreal, Canada, May 15-16, (afternoons).

The American Laryngological, Rhinological and Otological Society, Inc., will hold its Annual Meeting at the Sheraton-Mt. Royal, Montreal, Canada, May 15-16-17 (mornings only).

Please make early plans to attend the 1956 Spring Meetings in Canada. Both the Seigniory Club and Montreal present most attractive features for you and your family. More information about the places will be given later.

Reservations at the Sheraton-Mt. Royal Hotel should be made early by addressing the Reservation Supervisor, 1455 Peel Street, Montreal, P. Q., Canada.

POSTGRADUATE COURSE IN OTOLARYNGOLOGY.

The Department of Postgraduate Medicine of the University of Michigan Medical School announces the Otolaryngology Conference to be given at the University Hospital, Ann Arbor, Michigan, on April 19, 20 and 21, 1956, under the direction of Dr. A. C. Furstenberg, Chairman of the Department of Otolaryngology at the University of Michigan Medical School.

Guest lecturers of national prominence, together with members of the staff of the Department of Otolaryngology, will participate in the program.

For further information, write to Dr. John M. Sheldon, Director, Department of Postgraduate Medicine, University Hospital, Ann Arbor, Michigan.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Arrangements have been completed for the joint meeting of the North Carolina Society of Eye, Ear, Nose, and Throat, and the South Carolina Society of Ophthalmology and Otolaryngology September 17, 18, 19, 1956. Headquarters will be the George Vanderbilt Hotel, Asheville, North Carolina.

An unusually attractive program has been arranged, and a large attendance is anticipated.

Asheville, North Carolina, is in the mountains of Western North Carolina, and is a particularly beautiful spot in this season of the year.

For further information write Roderick Macdonald, M. D., Sec. and Treas., 330 East Main Street, Rock Hill, S. C.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

PROGRAM 1956

March 12 - 14, 1956

THE DALLAS SOUTHERN CLINICAL SOCIETY

Joint Meeting Fort Worth E.E.N.T. and Dallas O. and O.

GUESTS: Ophthalmology:
Dr. Trygve Gundersen
Dr. Jack S. Guyton

Otolaryngology:
Dr. Franz Altmann
Dr. Paul J. Moses

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Wm. J. McNally, 1509 Sherbrooke St., West Montreal 25, Canada.
Vice-President: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Secretary-Treasurer: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Seigniory Club, Ottawa, Canada, May, 1956.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Bernard J. McMahon, 8230 Forsyth Blvd., Clayton 24, Mo.
First Vice-President: Robert L. Goodale, 330 Dartmouth St., Boston, Mass.
Second Vice-President: Paul H. Holinger, 700 North Michigan Ave., Chicago 11, Ill.
Secretary: Harry P. Schenck, 326 South 19th St., Philadelphia 3, Pa.
Treasurer: Fred W. Nixon, 1027 Rose Building, Cleveland, Ohio.
Librarian, Historian and Editor: Edwin N. Broyles, 1100 North Charles St., Baltimore, Md.
Meeting: Mount Royal Hotel, Montreal, Canada, May, 1956.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Dean M. Lierle, Iowa City, Iowa.
President-Elect: Dr. Percy Ireland, Toronto, Canada.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Mount Royal Hotel, Montreal, Canada, May, 1956.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: John R. Lindsay, M.D., Chicago, Ill.
Vice-Chairman: James W. McLaurin, M.D., Baton Rouge, La.
Secretary: Hugh A. Kuhn, M.D., Hammond, Ind.
Representative to Scientific Exhibit: Walter Heck, M.D., San Francisco, Calif.
Section Delegate: Gordon Harkness, M.D., Davenport, Iowa.
Alternate Delegate: Dean Lierle, M.D., Iowa City, Iowa.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Daniel S. Cunning, 115 East 65th St., New York 21, N. Y.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia, Pa.
Meeting: Sheraton Mount Royal Hotel, Montreal, Canada, May 15-16, 1956 (afternoons only).

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill., October, 1956.

THE AMERICAN RHINOLOGIC SOCIETY

President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. James Chessen, 1829 High St., Denver, Colo.
Annual Clinical Session: Illinois Masonic Hospital, Chicago, Illinois, October, 1956.
Annual Meeting: Palmer House, Chicago, Illinois, October, 1956.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. D. M. Lierle, University Hospital, Iowa City, Iowa.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Palmer House, Chicago, Ill., October, 1956.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Joseph Gilbert, 111 E. 61st St., New York, N. Y.
Vice-President: Dr. Kenneth Hinderer, 402 Medical Arts Bldg., Pittsburgh, Pa.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16, N. Y.
Treasurer: Dr. Armand L. Caron, 36 Pleasant St., Worchester, Mass.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. J. M. Tato, Azcuenaga 235, Buenos Aires, Argentina.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
General Secretary: Dr. C. E. Muñoz MacCormick, P. O. Box 9111, Santurce 29, Puerto Rico.
Meeting: Fifth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: April 8-12, 1956, San Juan, Puerto Rico.
President: Dr. J. H. Font, Medical Arts Bldg., San Juan, P. R.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis, Mo.
General Secretary: Dr. Paul Holinger, 700 No. Michigan Ave., Chicago (11), Ill.
Meeting: Statler Hotel, Washington, D. C., May 5 - 10, 1957.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. William J. Hitschler.
Vice-President: Dr. Chevalier L. Jackson.
Treasurer: Dr. John J. O'Keefe.
Secretary: Dr. Joseph P. Atkins.
Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. Thomas F. Furlong, Jr., Dr. William A. Lell, Dr. Harry P. Schenck, Dr. Benjamin H. Shuster, ex-officio.

BALTIMORE NOSE AND THROAT SOCIETY

Chairman: Dr. Walter E. Loch, 1039 No. Calvert St., Baltimore, Maryland.
Secretary-Treasurer: Dr. Theodore A. Schwartz.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

President: Paul H. Holinger, 700 No. Michigan, Chicago 11, Ill.
Vice-President: Dr. Jack Allan Weiss, 109 No. Wabash Ave., Chicago 3, Ill.
Secretary-Treasurer: Dr. Stanton A. Friedberg, 122 So. Michigan Ave., Chicago, Ill.
Meeting: First Monday of each Month, October through May.

OTOSCLEROSIS STUDY GROUP.

President: Dr. Gordon D. Hoople, 1100 East Genesee St., Syracuse, N. Y.
Secretary: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Meeting: Palmer House, Chicago, Ill., October, 1956.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. J. L. Levine.
Vice-Chairman: Dr. Russell Page.
Secretary: Dr. James J. McFarland.
Treasurer: Dr. Edward M. O'Brien.
Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Frederick A. Holden, Medical Arts Building, Baltimore 1, Maryland.
Chairman-Elect: Dr. Sherman B. Forbes, 706 Franklin Street, Tampa 2, Florida.
Vice-Chairman: Dr. William J. G. Davis, 1632 K Street, N. W., Washington 6, D. C.
Secretary: Dr. G. Slaughter Fitz-Hugh, 104 East Market Street, Charlottesville, Virginia.

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Howard L. Mitchell, Lexington, Va.
President-Elect: Dr. L. Benjamin Sheppard, 301 Medical Arts Building, Richmond 19, Va.
Vice-President: Dr. Edgar Childrey, Jr., Professional Building, Richmond, Va.
Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Va.
Annual Meeting: May 26 - June 2, 1956.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. W. L. Hughes, Lamar Life Bldg., Jackson, Miss.
Vice-President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. J. C. Peele, Kinston Clinic, Kinston, N. C.

Vice-President: Dr. George E. Bradford, Winston-Salem, N. C.

Secretary-Treasurer: Dr. J. D. Stratton, 1012 Kings Drive, Charlotte 7, N. C.

Meeting: George Vanderbilt Hotel, Asheville, N. C., Sept. 16-19, 1956.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Norman Eaddy, Sumter, S. Car.

Vice-President: Dr. J. H. Gressette, Orangeburg, S. Car.

Secretary-Treasurer: Dr. Roderick Macdonald, 333 East Main St., Rock Hill, S. Car.

Meeting jointly with the North Carolina Eye, Ear, Nose and Throat Society, George Vanderbilt Hotel, Asheville, N. Car., Sept. 17-18-19, 1956.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Chas. C. Grace, 145 King St., St. Augustine, Fla.

President-Elect: Dr. Jos. W. Taylor, 706 Franklin St., Tampa, Fla.

Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

THE GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. A. R. Hollender, 605 Lincoln Road, Miami Beach, Fla.

Vice-President: Dr. Curtis D. Benton, Jr.

Secretary: Dr. James H. Mendel, Jr., 7241 Red Road, Miami, Fla.

Meeting: Quarterly, at Seven Seas Restaurant, February, May, October, and December.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Edwin G. Grafton.

Vice-President: Dr. L. A. Furchgott.

Secretary-Treasurer: Dr. Hal W. Maxwell.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Robert A. Norene, M.D.

Secretary-Treasurer: Leland R. House, M.D.

Chairman of Section on Ophthalmology: Wendell C. Irvine, M.D.

Secretary of Section on Ophthalmology: Stephen J. Popovich, M.D.

Chairman of Section on Otolaryngology: Herschel H. Burston, M.D.

Secretary of Section on Otolaryngology: Ross A. Goodcell, M.D.

Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles, California.

Time: 6:00 P.M., first Monday of each month from September to June inclusive—Otolaryngology Section. 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Lester T. Jones, M.D., 624 Medical Arts Bldg., Portland (5), Oregon.
Secretary-Treasurer: Dr. John F. Tolan, 3419 47th Ave., Seattle (5), Wash.
Meeting: April 15-19, 1956.

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Hon. Chairman: Dr. Isaac H. Jones, 635 S. Westlake Ave., Los Angeles 57, Calif.
Chairman: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.
Treasurer: Dr. Norman Jesberg, 500 So. Lucas Ave., Los Angeles 17, Calif.
Otolaryngology: Dr. Russell M. Decker, 65 N. Madison Ave., Pasadena 1, Calif.
Ophthalmology: Dr. Warren A. Wilson, 1930 Wilshire Blvd., Los Angeles 57, Calif.

Mid-Winter Clinical Convention annually, the last two weeks in January at Los Angeles, Calif.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

CANADIAN OTOLARYNGOLOGICAL SOCIETY
SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President: Dr. G. S. Gorrell, 110 Medical Arts Bldg., Calgary, Alta.
Secretary: Dr. G. Arnold Henry, 170 St. George St., Toronto, Ontario.
Meeting: June 6-7, 1956, Chateau Frontenac, Quebec, Canada.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Antonio da Costa Quinta, Lisbon, Portugal.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting:

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting: Lima, Peru, 1957.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villiers.
Vicepresidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salvador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

SOCIEDAD CUBANA DE OTO-LARINGOLOGIA.

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Vice-President: Dr. Jorge de Cárdenas.
Secretary: Dr. Pablo Hernandez.

SOCIEDAD DE ESTUDIOS CLINICOS DE LA HABANA

Presidente: Dr. Frank Canosa Lorenzo.
Vice-Presidente: Dr. Julio Sanguily.
Secretario: Dr. Juan Portuondo de Castro.
Tesorero: Dr. Luis Ortega Verdes.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBolde, Mexico, D. F.
Vice-President: Dr. M. Gonzalez Ulloa, Mexico, D. F.
Secretary: Dr. Juan de Dios Peza, Mexico, D. F.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretary of the Interior: Prof. Dr. Atilio Viale del Carril.
Secretary of Exterior: Dr. Aldo G. Remorino.
Secretary Treasury: Prof. Dr. Antonio Carrascosa.
Pro-Secretary of the Interior: Prof. Dr. Carlos P. Mercandino.
Pro-Secretary of the Exterior: Prof. Dr. Jaime A. del Sel.
Pro-Secretary of the Treasury: Dr. Jorge Zubizarreta.

BUENOS AIRES CLUB OTOLARINGOLOGICO

Presidente: Dr. K. Segre.
Vice-Presidente: Dr. A. P. Belou.
Secretario: Dr. S. A. Aranz.
Pro-Secretario: Dr. J. M. Tato.
Tesorero: Dr. F. Games.
Pro-Tesorero: Dr. J. A. Bello.

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLOMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix E. Lozano.
Tesorero: Dr. Mario Arenas A.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
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A STUDY OF THE ROLE OF CERTAIN FACTORS IN THE DEVELOPMENT OF SPEECH AFTER LARYNGECTOMY: 1. TYPE OF OPERATION; 2. SITE OF PSEUDOGLOTTIS; 3. COORDINATION OF SPEECH WITH RESPIRATION.[†]

Part 1: Type of Operation.

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In the literature of the last 25 years, it is evident that surgeons performing laryngectomies have become increasingly interested in various aspects of post-laryngectomy speech. The severity of the psychological, social and economic penalties

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[†] This paper, presented in three parts, is based on the research associated with "A Study of the Role of Three Factors in the Development of Speech after Laryngectomy: Type of Operation, Site of Pseudoglottis, and Coordination of Speech with Respiration," an unpublished Ph.D. dissertation (Northwestern University, 1954) by Evelyn Y. Robe. Subsequent issues of The Laryngoscope will carry Parts 2 and 3.

The research for the study was undertaken at the suggestion of Dr. Chevalier L. Jackson, Philadelphia, and was supported by a grant from the Illinois Division of the American Cancer Society.

Credit should be given to Mr. William W. Waldrop, Director, Speech and Hearing Rehabilitation Service, St. Luke's Hospital, for his advice and assistance in this study.

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resulting from loss of voice is repeatedly emphasized, as wel as is the importance to the laryngectomized person, of providing a means of audible communication as soon as possible

Although esophageal voice, the substitute voice which utilizes the patient's own pseudoglottis, is generally considered far more natural and desirable than that produced with the aid of mechanical devices, not all laryngectomized patients are able to develop it. More information is needed with regard to factors explaining why some patients successfully learn to use esophageal voice while others do not. Psychologica reasons for failure have been emphasized in the literature while factors such as the influence of anatomical or physiological changes resulting from the particular type of laryngectomy have received considerably less attention Psychological emphasis may be warranted,[1] but the lack o adequate data at this time prevents an evaluation of the relative importance of these observations.

One purpose of the present study was to discover the effect of the following factors in the success or failure of laryngectomized patients to develop esophageal voice:

1. Type of operation: narrow field laryngectomy, modified wide field laryngectomy, wide field laryngectomy, and laryngectomy with radical neck dissection.
2. Pre-operative esophageal speech training.
3. Length of post-operative esophageal speech training.

Relatively few investigators have expressed opinions concerning the influence which operative technique may have upon the development of substitute voice. Since no attempt to bring these opinions together for examination and comparison has been found, it is appropriate that they be reviewed here.

Stern, one of the earliest investigators in the field, expressed the belief, as quoted by Gatewood, that, "The glottis location probably depends upon the conditions created in the operation, and is of great importance to the success of speech." Gatewood reported also that Gutzman, another early writer on post-laryngectomy speech, believed that the voice is largely dependent upon the individual anatomic construction of the upper esophagus and hypopharynx.[3]

Gatewood expressed the opinion that, "total extirpation of the epiglottis has shown no derogatory effect upon voice development, nor has the voice shown any superior quality in those whose epiglottis was not removed."[5]

In 1934, Kallen wrote:

"The surgeon should be interested in preparing the patient as favorably as possible for the phonetician. He should save as much tissue as is safely possible. In the desire to be absolutely sure that no diseased portion remains he often removes demonstrably sound tissue, such as muscular tags, folds of mucous membrane, etc. Such removal is to be regretted. It should be performed only when doubt as to the health of the tissue makes it imperative. Every fold of mucous membrane, every favorably placed cicatricial band, every muscle or muscular remnant, may serve as the basis for the development of a pseudoglottis . . . Many a striking case of pseudo-voice owes its development to the hand of the watchful surgeon.[7]."

More specifically, Kallen recommended that:

"The surgeon should exercise the greatest care during the operation to spare the fibers of the cricopharyngeus muscle. Much of the damage which it sometimes receives can be avoided by dissecting it away from the cricoid cartilage. Not only is it of the utmost importance that this structure escape all injury during operation, but the integrity of its innervation must also be preserved. Lacking that, it cannot properly function as a pseudoglottis. The esophageal branches of the recurrent laryngeal nerve must be spared as much as possible to retain the external branches of the superior laryngeal nerve, though these are also involved in the innervation of the cricopharyngeus muscle."

"Saving the esophageal orifice from surgical harm is an essential precaution. In addition, it is important to protect the sternohyoid and *thyrohyoid* muscles. These muscles pull the larynx upward and forward, lifting it away from the posterior wall of the pharynx, thereby relaxing the tonic closure effected by the cricopharyngeus muscle—an important movement in the training of esophageal speech. Finally, the phonetician would ask the surgeon to sew the sternohyoid and the thyrohyoid muscles to the anterior wall of the pharynx, approximately at the level of the mouth of the esophagus. This facilitates the mechanism of aspiration."[7]

Other proponents of surgical procedures which will conserve the cricopharyngeus and preserve the nerve supply to the esophagus are Guttman, Levin, Morrison, Gatewood, Luchsinger, Arnold and Mason.

In 1935, Guttman reviewed the cases of a number of patients who could not acquire a substitute voice. Every one was found to have an esophageal introitus which was always patent. The overly relaxed condition, Guttman believed, was due to "too extensive coaptation or the severance of the motor nerves at the time of laryngectomy." As a result of these findings, he reports, "we have used a great deal of care in retaining as much of the cricopharyngeus muscle as possible in resecting the nerve supply to the esophagus."[4]

Levin, a laryngeal surgeon who instructs his patients in post-laryngectomy speech, agrees with Kallen that not only the cricopharyngeus muscle and esophageal nerve supply but also the sternothyroid and thyrohyoid muscles should be preserved. As did Kallen, he advocates, "suturing these muscles to the anterior wall of the pharynx approximately at the level of the mouth of the esophagus, because they assist in the aspiration of air into the esophagus."[9]

"Laryngectomy by the technique of Crowe and Broyles, and later by that of Gabriel Tucker," Levin states, "tends to conserve these structures very efficiently."

In 1952, Levin again urged preservation of the upper esophageal sphincter and nerve supply and also that an effort be made to create an ample hypopharyngeal pouch. This is because "a pharyngeal or upper esophageal stenosis is a serious complication in future speech training." He is also of the opinion that:

"There is still a need for technical modifications to facilitate post-operative speech development. My operative efforts now include more consideration for the anatomic structures that may favorably affect future esophageal speech requirements. Further experience in this regard is needed."[9]

Morrison (1941) expressed the belief that the achievement of a stronger and more easily produced pseudovoice would be quite certain when the musculature, nerve and blood supply of the hypopharynx and upper esophagus are preserved as fully as possible. This expectation was based on the consideration that at least part of the musculature of the upper esophagus is striated and under voluntary control, and is supplied by fibers of the recurrent laryngeal nerves.[12]

Gatewood (1944) suggested that better sphincteric action of the cricopharyngeus muscle might result in a vertical closure of the esophageal opening. With regard to the epiglottis, he commented that total removal had shown no derogatory effect upon voice development, while those in whom the epiglottis remained had not developed voices which were superior in quality. In accord with Levin and Kallen, Gatewood encouraged preservation of the anterior ribbon muscles in order to avoid impairing the antagonistic action of the sphincter fibers of the esophageal orifice.[2]

Luchsinger and Arnold in Vienna (1949) and Mason in England (1950) added their recommendations for operative technique which would preserve the cricopharyngeus muscle, the esophageal nerve supply and the strap muscles.[10,11]

Jackson and Jackson, in *The Larynx and Its Diseases* state the following "with a view to aiding articulate speech without a larynx":

a. The tip of the epiglottis may be allowed to remain.

b. The cricopharyngeus muscle may be left uninjured; it often serves well for the new pharyngeal glottis that produces the vibratory or fricative sound that will serve for speech when modified by the molds.

c. Utmost care must be taken to avoid a narrow pharynx, because the larger the pharynx, the easier it is for the patient to gulp air.

d. No tissue should be unnecessarily sacrificed.

e. The trunk and external branch of the superior laryngeal nerve should be left uninjured if possible.

f. The external lifting muscles of the larynx should be left attached with a view to lifting the lower pharynx upwards and forwards. This helps to open the esophagus.[6]

The possibility of the deliberate creation of a fistula during or following laryngectomy as an aid to speech development, has been discussed by Guttman and by Jackson.

Guttman was motivated by the experience of a laryngectomized patient, who, because he objected to an artificial larynx, had taken a heated ice pick and passed it through the trachea to the hypopharynx several times, thus forming a permanent fistula. He was able to use the fistula to provide an air supply for an excellent substitute voice.[4]

Guttman's procedure was to insert a needle electrode through the tracheal stoma and up behind the remnants of the epiglottis so that the opening would be partly protected from the ingress of food and water to the trachea. Within two weeks, the electrocoagulated tract usually developed a definite fistula. After formation of the fistula, speech can be taught very easily:

> "The patient is taught to block the tracheal stoma with his finger or thumb and then force the air up along the fistulous tract. He easily learns to modulate the resulting tones into speech. By varying the tension on the fistula, by more or less flexion of the head, he can also vary the pitch to some extent and so produce a more pleasing and less monotonous voice."[4]

Jackson and Jackson included as another suggestion that a fistula be permitted to remain. The procedure recommended for this was very similar to Guttman's. They, too, state that the best place for the fistula would be as close as possible to the tip of the epiglottis, thus helping to divert food and fluid from entering the trachea; moreover, they advocated:

> "The opening after it is epithelialized need not be larger than 2 mm. in diameter, about the thickness of a straw; an orifice of this size would be amply sufficient to allow air to leak upward and will be closed by the collapse of its edges,

if not by a fold or by the tip of the epiglottis as the food comes downward. If after a time the patient is unable to get along with such a fistula because of the leaking of food and strangling, the fistula can be closed by a small plastic procedure".[6]

The effect which combined laryngectomy and radical neck dissection might have on later development of a substitute voice should also be considered. Unfortunately, very little information on this aspect of the subject has been published. Ogura expressed the opinion that substitute voice is possible in most patients who have had the combined operation.[13]

It should be noted that all those whose opinions were cited have stressed strongly that excision of malignant tissue must always be the primary consideration in laryngectomy, and that conservation of tissue as an aid to later voice acquisition, although desirable, is necessarily a secondary concern.

The scarcity of information about the type of laryngectomy and the related speech training led to a direct study of reports and patients.

Thirty-two persons who had been laryngectomized within the preceding five year period were selected as subjects for this section of the study. Data concerning the type of laryngectomy, post-operative recovery factors, and pre-operative speech training were obtained from the case histories and operative reports provided by the surgeons who performed the laryngectomies. Information regarding the amount of post-operative speech training which the subjects had received was obtained from their teachers.

After each subject was interviewed several times and observed in a variety of speaking situations, his speech fluency was evaluated in terms of the scale shown in Table V. This scale was a revision of one developed earlier in a preliminary study.[1]

The information from the several sources was classified as shown in Table I under the following headings: *1*—Sex; *2*—Age at time of laryngectomy; *3*—Type of laryngectomy; *4*—Postoperative recovery factors which might have influenced later speech development; *5*—Pre-operative speech

TABLE I

Subject	Sex	Age at Time of Laryngectomy	Type of Laryngectomy	Post-operative Recovery Factors Which Might Have Influenced Speech Development	Pre-Operative Speech Training	Post-Operative Speech Training (Number of Lessons)	Present Evaluation of Speech Fluency (See Table V)	Comments
1	M	27	Laryngectomy and Right Radical Neck Dissection	....	....	17 Class	c	
2	M	48	Wide Field	....	....	4 Individual	g	
3	F	32	Laryngectomy and Left Radical Neck Dissection	....	....	2 Individual 1 Class	g	Prefers to use electro-larynx because of gastric discomfort accompanying esophageal voice production.
4	M	51	Modified Narrow Field	....	....	6 Individual	d	
5	M	39	Narrow Field	....	....	8 Individual	g	
6	M	48	Modified Narrow Field	....	....	4 Class	g	
7	M	57	Modified Narrow Field	....	....	3 Individual 1 Class	g	
8	M	56	Wide Field	....	....	16 Individual	f	
9	M	48	Narrow Field	....	....	2 Individual	g	
10	M	71	Modified Narrow Field	Some Swallowing Difficulty	....	3 Individual 10 Class	f	Has Hearing Loss
11	M	60	Partial Laryngectomy (Epiglottis and Cricoid Remain)	Infection About Cricoid Cartilage	....	5 Individual	d	
12	M	60	Narrow Field	....	....	9 Individual	f	
13	F	52	Wide Field and Thyroidectomy	....	....	8 Individual	g	
14	M	47	Narrow Field	....	....	16 Individual	f	
15	M	53	Narrow Field	....	....	1 Individual	g	
16	M	54	Narrow Field	....	....	9 Individual	f	

TABLE I — Continued

Subject	Sex	Age at Time of Laryngectomy	Type of Laryngectomy	Post operative Recovery Factors Which Might Have Influenced Speech Development	Pre-Operative Speech Training	Post-Operative Speech Training (Number of Lessons)	Present Evaluation of Speech Fluency (See Table V)	Comments
17	M	51	Wide Field	Fistula	...	10 Individual	g	
18	M	51	Narrow Field			5 Class	c	
19	M	54	Narrow Field			8 Individual	g	
20	M	60	Wide Field			3 Class	f	
21	M	49	Narrow Field			2 Individual	g	
22	M	68	Wide Field		1 Individual	5 Individual	g	
23	M	57	Narrow Field	Fistula Postponed Speech Training		2 Individual 7 Class	e	
24	M	48	Narrow Field			8 Individual	g	
25	M	59	Wide Field			9 Individual	g	
26	M	56	Narrow Field		–	1 Individual 8 Class	g	
27	M	58	Narrow Field	...		4 Individual	g	
28	M	70	Narrow Field	Fistula		2 Individual 6 Class	g	
29	M	53	Narrow Field			1 Individual 7 Class	g	
30	M	64	Wide Field	–		4 Individual	a	Became discouraged Will not continue lessons
31	F	29	Modified Wide Field	–		5 Individual 2 Class	f	
32	M	70	Modified Wide Field			5 Individual 2 Class	b	

training; *6*—Post-operative speech training; *7*—Speech evaluation; *8*—Comments.

Only significant characteristics of the group and simple relationships between type of laryngectomy were considered since the probability of unknown variables made detailed statistical analyses unwarranted.

Of the 32 cases included in this section of the study, 29 were male and three were female. The preponderance of males bears out the findings of other investigators who have reported sex ratios varying from nine to one to 14 to one.[5]

Review of the ages of the subjects at the time of laryngectomy reveals a range of 27 to 71 years, with the highest incidence in the 50-60 year group. This also agrees with the figures published by other investigators.[14] A classification of incidence in the various age groups appears in Table II.

TABLE II.

INCIDENCE OF LARYNGECTOMY RELATED TO AGE IN EXPERIMENTAL GROUP.

20-30	30-40	40-50	50-60	60-70	70-80
27	32	47	51	60	70
29	39	48	51	60	70
		48	51	60	71
		48	52	64	
		49	53	68	
			53		
			54		
			54		
			56		
			56		
			57		
			57		
			58		
			59		
2	2	6	14	5	3

Classification of the group according to type of laryngectomy is shown in Table III.

Post-operative complications, such as infection or fistula occurred in five cases but had no adverse effect in at least three who developed excellent speech.

TABLE III.

INCIDENCE OF TYPE OF LARYNGECTOMY IN EXPERIMENTAL GROUP.

Laryngectomy and Radical Neck	Wide Field	Narrow Field	Modified Narrow Field	Partial Laryngectomy
2	8	17	4	1

Only one subject of the group received pre-operative speech training.

Table IV indicates that the average number of speech lessons for the patients who developed fluent speech was: Class, 9; Individual, 7; and a combination of Class and Individual, 8.

TABLE IV.

CLASSIFICATION OF AMOUNT OF GROUP AND INDIVIDUAL POST-LARYNGECTOMY SPEECH INSTRUCTION.

			Individual and Class	
	Class	Individual	Individual	Class
	3	1	2	1
	4	2	3	1
	5	2	3	10
	17	4	2	7
		4	1	8
		4	2	6
		5	1	7
		5	5	2
		6	5	2
		8		
		8		
		8		
		8		
		9		
		9		
		9		
		10		
		16		
		16		
Total number of subjects	4	19	9	
Average number of lessons	9	7	8*	

* Individual and group combined.

Speech fluency ratings (Table V) ranged from the lowest on the scale (no sounds produced), to the highest (fluent, non-hesitant speech). Eighteen of the 32 subjects received the highest rating. The number of speech lessons which the person had had did not seem to be related to speech fluency. That is, some were fluent after one lesson, others needed more instruction.

TABLE V.

CLASSIFICATION OF SUBJECTS ACCORDING TO SPEECH FLUENCY.

a.	No sounds produced	1
b.	Partial control; single sounds under fair control	1
c.	Simple words produced	2
d.	Combines 2-3 words	2
e.	Some sentences used	1
f.	Sentences consistently used	7
g.	Fluent, non-hesitant speech	18

With regard to the relation of the type of laryngectomy, the following observations were made on the ability of the subjects to develop speech:

Seventeen of the subjects had had a narrow field laryngectomy, in which the thyrohyoid and sternothyroid muscles are usually preserved. The fact that 10 of the 17 received the highest rating on the fluency scale, and that three others were given the second highest rating, suggests that a relationship between the type of laryngectomy and the later ability to develop substitute voice may have existed in this group. That substitute voice does not always depend on the presence of the strap muscles, however, is evident from the number of those who developed fluent speech following wide field laryngectomy, and from the fact that one of the two who had had laryngectomy with radical neck dissection was able to develop fluent speech.

It will be noted that the majority of subjects available for these studies had had a narrow field operation. This does not reflect conservatism in surgery but is probably due to the fact that for the most part, they were referred as private patients. The lesions were generally operated upon earlier than in a similar group that might have been selected from a large charity service; furthermore, the general intelligence level and educational background were higher than those of a similar group that might have been obtained from a large charity service. It is for these reasons, rather than that the majority of the subjects had had a narrow field laryngectomy that the type of operation is not considered significant; furthermore, since many patients developed a satisfactory voice following wide field operation or radical neck dissection in association with laryngectomy, preservation of the thyrohyoid and sternothyroid muscles is likewise not considered too significant.

Mention should be made of the five subjects who had had a modified narrow field laryngectomy and the one who had had a modified wide field laryngectomy. The modification in both types consists essentially of resection of the middle section of the hyoid bone and tapering of the pharynx downward. It is possible that this type of surgery should prove a positive aid to post-operative speech development when the pseudoglottis is formed at the pharyngoesophageal juncture. For this reason, it is unfortunate that more cases which had undergone this type of surgery were not available for the study Indications for this modified surgery are infrequently seen. The fact that three of the group experienced difficulty in learning to use a substitute voice largely because of the infirmities of age, such as hearing loss, make it impossible to evaluate the direct effect of the operation on later speech. The remaining three of the group who were free of the problems just mentioned developed fluent speech after fewer than the average number of lessons.

It was not always possible to ascertain from the operative reports the exact percentage of the cricopharyngeus that was preserved; nor is it possible to estimate the amount of actual functioning muscle which is present after the healing process has been completed. No conclusions can be drawn, therefore, in regard to the role it may have played in the post-operative speech development of this group.

SUMMARY.

Data concerning the type of laryngectomy, post-operative recovery factors, and the amount and kind of pre-operative and post-operative speech training, were obtained from the case histories and operative reports of 32 persons who were laryngectomized within the preceding five-year period. The information was analyzed to determine possible relationships between the several factors and the success or failure to develop substitute voice following laryngectomy.

Results of the analysis indicated that the types of surgery represented in this study did not in themselves appear to determine the relative excellence of the speech result nor the amount of speech training necessary to achieve satisfactory post-laryngectomy speech.

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DISEASES OF THE FACIAL NERVE.*†

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An anatomic and clinical study of the facial nerve may at first glance appear to be an academic exercise; however, as the number of articles in the current literature will testify, interest in the facial nerve is not a static one. Recent advances in anatomical concepts, diagnostic procedures, and medical and surgical management of diseases affecting this cranial nerve are considerable and worthy of renewed consideration. From a practical point of view problems relating to the facial nerve are constantly recurring in every otological clinic or office. We would like to emphasize the plea of Cody[1] that the otologist familiarize himself with the disorders of the facial nerve. By interest and training the otologist is in a favorable position to manage competently all types of facial nerve disease.

In attempting to present a concise study of facial nerve disease we must confess we have been thwarted. It is not possible to do justice to the voluminous, often controversial, literature on the subject in the time allotted; therefore only portions of the study will be presented.

In Fig. 1, we show a general outline of the subject to be presented, and then a series of photographs of patients with facial palsies of various causations.

The anatomic features of the facial nerve is presented in diagrammatic form in Figs. 2-7, in which the main course of the nerve and its two principal branches, the supra nuclear pathways and the parotid-facial portion of the nerve, are illustrated.

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* Read at the Meeting of the South[illegible] Rhinological and Otological Society, I[illegible]

† From the Dept. of Otolaryngology,

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Fig. 1.

GENERAL OUTLINE.

- A. Anatomic Features
    1. Facial nerve and principal branches
    2. Chorda Tympani nerve—recent studies
- B. Facial Paralysis
    1. Classification
    2. Etiology and Diagnosis
    3. Electrodiagnostic Methods
    4. Bell's Palsy
    5. Otogenous Paralysis
    6. Paralysis following Cranial Trauma
    7. Congenital Facial Paralysis
    8. Facial Paralysis associated with Poliomyelitis
    9. Facial Paralysis in Systemic Disease
    10. Facial Paralysis in Uveo-parotid Syndrome
    11. Hemifacial Spasm
    12. Syndrome of Crocodile Tears
    13. Medical Management
    14. Surgical Treatment
        - a. Anastomosis
        - b. Plastic Surgery
    15. Sequelae of Facial Paralysis
- C. Tumors of the Facial Nerve
- D. Melkersson's Syndrome
- E. Tic Douloureux of the Nervus Intermedius
- F. Herpes Zoster of the Geniculate Ganglion
- G. Geniculate Neuralgia
- H. Discussion of the Surgical Aspects of the Parotid Portion

A. *Supranuclear Pathways*[2] (see Fig. 2).

The supranuclear fibers concerned with voluntary facial movements originate in the lower portion of the precentral gyrus and proceed downward to the facial nucleus on the opposite side. The supranuclear fibers supplying the upper portion of the face arise from both cortices. The fibers controlling emotional facial movements, arising probably in the thalamus, do not run in the same pathways as those fibers controlling voluntary movements.

B. *Infranuclear Pathways* (see Fig. 3).

The nucleus of the facial nerve lies in the ventral part of the tegmentum of the pons between the olivary nucleus and

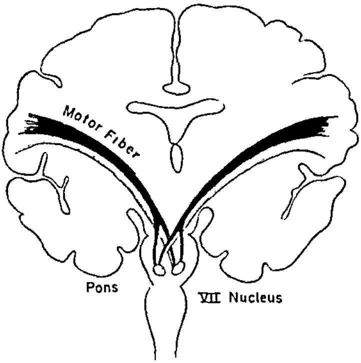

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Fig 2 Supra nuclear pathways

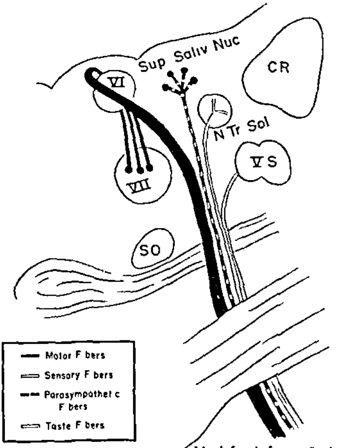

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Fig 3 Area of nucleus of facial nerve

restiform body. From this origin the facial fibers pursue a curved course through the pons, looping about the abducens nucleus, and emerging from the brain at the lower border of the pons between the olive and the inferior peduncle. It then passes laterally upward and forward across the subarachnoid space in company with its sensory component, the nervus intermedius, and the VIIIth cranial nerve, to the internal auditory meatus. Within the meatus the nervus intermedius joins the motor portion of the facial, and the common trunk enters the facial canal.

C. *Intrapetrous Pathway.*

The course of the facial nerve through the petrous bone has been divided into four parts:[3]

1. The portion in the internal acoustic meatus.

2. The lateral segment from the fundus of the internal acoustic meatus to the geniculate ganglion.

3. The horizontal segment along the medial wall of the tympanic cavity.

4. The vertical portion extending downward to the stylomastoid foramen.

1. Within the internal auditory meatus, the facial nerve above and the acoustic nerve below, divide at the fundus of the meatus where the facial nerve enters the facial canal. In the meatus a communicating branch between the two nerves is present.

2. The lateral or labyrinthine segment traverses the upper portion of the bony labyrinth between the cochlea and vestibule to a point where it bends sharply laterally and posteriorly to reach the internal wall of the middle ear. The geniculate ganglion, the origin of the sensory root of the facial nerve, is located just medial to the external genu of the facial nerve. Here the following branches of the facial nerve are given off:

a. The greater superficial petrosal nerve.

b. A communicating branch to the lesser superficial petrosal nerve which joins the tympanic nerve from the tympanic plexus to connect with the otic ganglion.

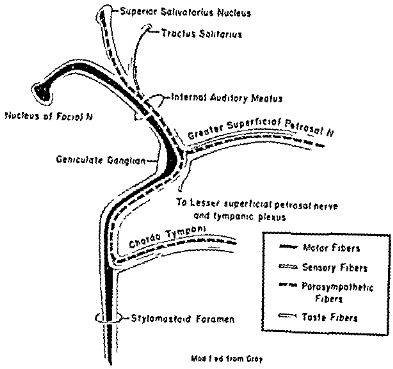
c. The external superficial petrosal nerve which joins the middle meningeal artery sympathetic plexus.

3. The horizontal or tympanal segment runs in a bony canal along the tympanic wall of the inner ear, starting just anterior to the process cochleariformis and ending in a broad curve downward behind the base of the pyramidal eminence, passing above the oval window and beneath the lateral semicircular canal. The branch for the stapedius muscle is given off as the facial nerve passes behind the pyramidal eminence.

4. In the vertical or mastoidal portion the nerve proceeds downward to emerge from the skull at the stylomastoid foramen. In this portion two branches are given off:

a. The chorda tympani nerve.

b. Communicating branches to the auricular branch of the vagus.



Modified from Grey

Fig. 4. Main course of facial nerve.

Kettel[4] and others have pointed out that occasionally the course of the vertical portion is abnormal, lying posterior to the tympano-mastoid line; the operator should be aware of these anatomical variations. Guild[5] has written of the natural absence of part of the bony wall of the facial canal. A gap in the continuity of the canal may be present in any part of its course.

D. *Parotid Pathways.*

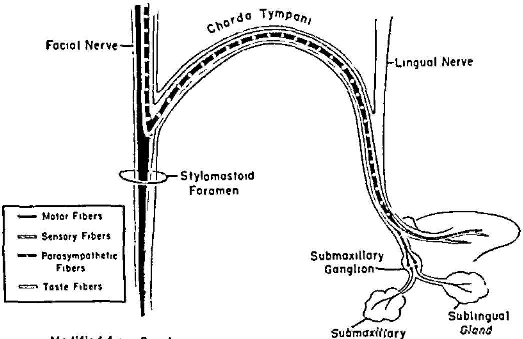
After emerging from the skull at the stylomastoid foramen, the peripheral portion of the facial nerve runs laterally and forward between the styloid and mastoid processes in loose areolar tissue to penetrate the posterior edge of the parotid gland. Shortly thereafter, the main trunk of the nerve divides into two main branches, the temporofacial and cervicofacial, which in turn separate into many radiating and anastomotic branches supplying chiefly motor innervation to the muscles of the face and scalp. These secondary branches may be divided into those supplying the temporal, zygomatic, buccal, mandibular, and cervical segments of the face. Between the stylomastoid foramen and the initial division of the main nerve trunk, a distance varying between 9.5 to 2.0 cm., filaments are given off to the skin and muscles of the occipital, postauricular, and subauricular regions. For practical and surgical reasons, it makes little difference as to the variations in the position of the nerve in relation to the parotid gland tissue and in its branchings; however, one should be familiar with these variants, as noted by such investigators as Hollinshead,[6] McWhorter,[7] McCormack,[8] State,[9] McKenzie,[10] and others. We have not been confused when the variations have been encountered in our surgical and cadaveric dissections. The facial nerve is difficult to dissect free only when closely incorporated in inflammatory, neoplastic or scar tissues.

The peripheral facial nerve not only is responsible for motor innervation of the facial muscles but also is the pathway for minor, cutaneous sensory fibers to the auricular area, probably sensory deeper (proprioceptive) fibers for the face, and secretory (preganglionic) fibers for the parotid gland tissue. The latter is by way of the IXth nerve, otic ganglion, and the auriculotemporal-facial connective fibers, as outlined by Reissner.

Figs. 5-6 illustrate the anatomical pathways of the two principal branches of the facial nerve:

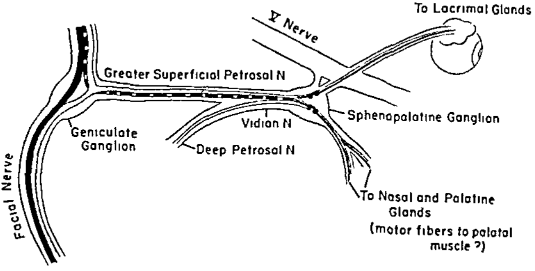
1. The greater superficial petrosal nerve, branching off at the site of the ganglion, passes anteriorly to enter the middle

cranial fossa. It then runs in a sulcus on the anterior surface of the petrous pyramid, passes beneath the semilunar ganglion and proceeds through the foramen lacerum. There it joins the deep petrosal from the internal carotid sympathetic plexus to form the nerve of the pterygoid canal and enters the trigeminal system at the sphenopalatine ganglion.



Modified from Grant

Fig. 5. Course of chorda tympani nerve.



Modified from Boyer and Gardner

Fig 6. Course of greater superficial petrosal nerve.

The greater superficial petrosal nerve is the motor root of the sphenopalatine ganglion; it contains preganglionic parasympathetic fibers from the nervus intermedius portion of the facial nerve and sensory fibers whose cells of origin are in the geniculate ganglion. This nerve, therefore, contributes post ganglionic vasodilator and secretory fibers to the nose and lacrimal gland, and some sensory fibers to the posterior portions of the nose, to a portion of the pharynx and hard and soft palates.

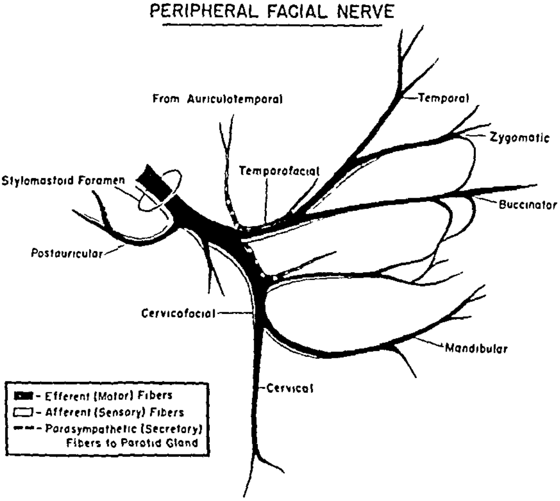


Fig. 7. Extracranial portion of facial nerve.

2. The chorda tympani nerve leaves the facial nerve about 4-6 mm. above the stylomastoid foramen to enter the iter chordae posterior. In this canal it runs a recurrent course upward and forward, entering the middle ear cavity beside the rib of the tympanic membrane, passing between the malleus and incus, and leaves the middle ear via the iter chordae anterior. The nerve then emerges from the skull through the petro tympanic fissure, and joins the lingual nerve at an acute angle at the side of the nasopharynx close to the root of the tongue.

The chorda tympani nerve is the pathway of taste fibers from the anterior two-thirds of the tongue. The nerve fibers from the tongue pass through the lingual and chorda tympani nerves to reach the geniculate ganglion. The central pathway from the ganglion is to the nucleus solitarius through the nervus intermedius, or via the greater superficial petrosal-trigeminal pathways, or possibly via the connections with the tympanic plexus and the IXth nerve.

The chorda tympani also carries intraoral sensory fibers and preganglionic motor fibers to the submaxillary ganglion, through which post ganglionic fibers reach the submaxillary and sublingual salivary glands.

There is an uncertain connection between the facial nerve and the otic ganglion. Preganglionic fibers from the facial nerve have been described which connect with the otic ganglion via the greater and lesser superficial petrosal nerves or fibers from the chorda tympani.

The sensory fibers of the facial nerve have been of great clinical and controversial interest. Apparently a great majority of the sensory fibers leave through the greater superficial petrosal and chorda tympani nerves. Some sensory fibers emerge with main motor branches of the nerve and provide deep sensibility of the face. The auricular branch of the facial nerve, together with the auricular branches of the IXth and Xth nerves supply innervation to the concavity of the concha, the posterior portion of the external auditory meatus, a portion of the external surface of the tympanic membrane and a small area on the posterior aspect of the auricle and adjacent mastoid area. This innervation is an important part of the concept of geniculate herpes zoster (see Fig. 8).

The blood supply of the facial nerve (mastoidal portion) is chiefly derived from the external carotid system via the stylomastoid branch of the posterior auricular artery and the superficial petrosal branch of the middle meningeal artery. The petrous segment is supplied by *1.* the tympanic branch of the internal maxillary artery; *2.* the ascending pharyngeal branch of the external carotid artery; *3.* branches of the middle meningeal artery and *4.* caroticotympanic branch of the internal carotid artery.

Recent interest in the chorda tympani nerve has resulted in part by the endaural surgical approach to temporal bone disease. Observations by various surgeons during fenestrations, experimental studies on the effects of stimulation of the chorda tympani, and an evaluation of the concept of Rosen[11] on the role of the chorda tympani nerve in tinnitus, vertigo and deafness, have shown generally that this nerve is concerned with taste on the anterior two-thirds of the tongue, and in pain.

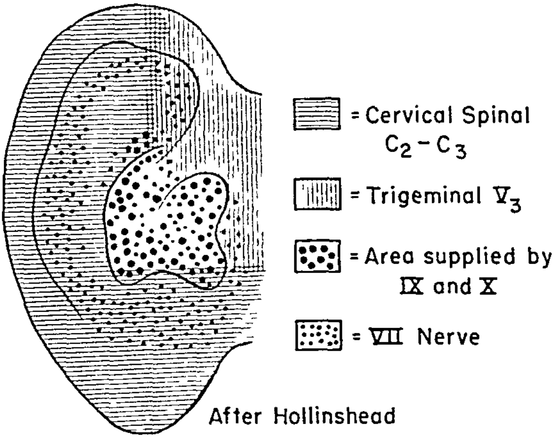


Fig. 8. Sensory nerve. Distribution to auricle.

Electrical stimulation of the chorda tympani (Frenckner and Preber,[12] Elliasson and Gisselsson[13]) produced pain and a metallic taste in the anterior part of the tongue. None of the primary taste sensations (salty, sour, sweet and bitter) or vertigo, or tinnitus could be obtained by stimulation of the chorda tympani nerve. Scheer[14] found that if the nerve is picked up, stretched and then avulsed there is a sharp pain that radiates down the neck and mandible. The patients have an annoying, bitter, metallic taste after the nerve is severed, lasting in two cases over eight months.

Costen, Clare and Bishop[15] point out that the chorda tympani nerve carries preganglionic fibers from the superior salivatory nucleus to the submaxillary ganglion and supply taste sensation to the anterior two-thirds of the tongue. Their observations indicate that the chorda tympani carries taste, touch and pain sensations to the tongue, and they emphasize that this nerve is essentially a sensory nerve with an incidental component for the special sense of taste.

The rationale of Rosen's[11] treatment of Meniere's disease by section of the chorda tympani nerve involves the anatomical connection between the nervus intermedius and the vestibular portion of the VIIIth nerve, and the close association of the tractus solitarius with the cochlear and vestibular nuclei. Section of the chorda tympani nerve and tympanosympathectomy as used by Rosen and a part of Frenckner's[16] technique in the surgical treatment of tinnitus aurium and Meniere's disease, is based on Schneider's[17] hypothesis of a double sonic system.

Lempert[18] believes that the chorda tympani nerve is concerned only with salivary gland secretion and sensory fibers of taste and is in no way involved in inner ear dysfunctions.

FACIAL PARALYSIS.

It has been stated that facial paralysis constitutes 75 per cent of lesions of the facial nerve. Cawthorne[19] has said that the facial nerve is more frequently paralyzed than any other motor nerve in the body, and has the longest course in a bony canal of any nerve. Ninety per cent of peripheral facial paralysis is caused by lesions in the temporal bone.

CLASSIFICATION.

I. Supra Nuclear Lesions:
    Intracranial tumor, vascular accident, etc

II Nuclear Lesion
    A Congenital nuclear aplasia
    B Pontine Lesions (Tumor, poliomyelitis, inflammations, syringobulbia, etc)

III Posterior Cranial Fossa Disease
    A Cerebello pontine angle tumors
    B Meningeal lesions
    C Tumors of Petrous Ridge

IV. Paralysis from Injury of Disease of Temporal Bone
    A Neurinomas
    B Fractures
    C. Inflammation
    D Surgical Injury

V Paralysis from Injury of Disease of the Extra Temporal Course:
    A. Parotid tumors
    B Birth injuries
    C Trauma
    D Uveo parotid syndrome

VI Bell's Palsy.

McCaskey's[20] classification is as follows:

1 Trauma
  a Operative
  b External violence
2 Infection
  a Cerebral abscess, encephalitis, meningitis, poliomyelitis
  b Herpes zoster
  c Acute and chronic otitis media
3 Exposure—Bell's Palsy
4 Neoplasm
5 General Systemic Disease
  a Diabetes
  b Syphilis
  c Cardio vascular disease
  d T B
  e Leukemia
6 Allergy (Milk)
7 Toxicosis—Alcohol
  Drugs

Etiology of Facial Paralysis.

An idea of the etiological factors in facial paralysis can be obtained from the following tabulations.

In Cawthorne's[21] series of 97 cases:

1 Operative Injury		32
Radical Mastoid	14	
Cortical Mastoid	11	
Labyrinth Operation	4	
Foreign Body .	3	
2 Bell's Palsy		26
3 Chronic Suppurative Otitis Media		15
4 War Injury .		11
5 Skull Fractures		4
6 New Growth .		3
7 Accident		2

In Cawthorne's[19] later series of 347 cases:

90% of lesions were within temporal bone
10% were intra or extracranial

Site and cause of lesion in 138 cases of intratemporal facial palsy found at operation were as follows:

A Tympanic Segment	
Injury	17 cases
Infection	9 cases
New Growth	2 cases
B Bend or Upper Vertical Segment	
Injury	28 cases
Infection	15 cases
New Growth	2 cases
C Lower Vertical Segment	
Bell's Palsy	62 cases
Injury	2 cases

In Cawthorne's[21] series of 325 cases reported in 1952 the following tabulation was made:

Intracranial—9 cases		Intratemporal —301 cases		Extracranial —15 cases	
New Growths	4	Bell's Palsy	205	New Growth	3
Disseminated Sclerosis	2	Injury	47	Injury	12
Poliomyelitis	3	Acute Otitis Media	26		
		Herpes	18		
		New Growth	5		

In Park and Watkins'[23] series of 500 cases:

87 2% were termed idiopathic
6% traumatic
6% infection
8% tumor

In a series of 60 cases of peripheral facial palsy recently tabulated at the University of Virginia Hospital we found:

Idiopathic	40%	Meningitis	1 7%
Traumatic	47%	Melkersson's	1 7%
Mastoiditis	4 7%	Glomus jugulare	1 7%
		Cerebellar pontine angle tumor	1 7%

DIAGNOSIS.

The diagnosis of facial paralysis is usually quite evident. The localization of the site of the lesion demands more careful study. A diagrammatic representation of the localizing factors using Tschiassny's[24, 25, 26] outline in part, is as follows:

Supranuclear Lesion·
Paralysis lower one third of face (orbicularis, corrugator supercillii, and frontals are not affected)
Associated weakness arm and leg usually same side
Absence of Bell's phenomenon
Emotional movements and reflexes not affected
Taste and lacrimation—normal

Nuclear Lesion
Facial monoplegia
May have associated VIth nerve paralysis
Lacrimation, salivation and taste not affected
Loss of reflexes and of emotional movements

Posterior Cranial Fossa
Facial monoplegia late
Associated auditory and vestibular signs
Taste frequently not affected
Associated Vth nerve signs (corneal anesthesia)
Associated intracranial pressure signs
Cerebellar signs

Temporal Bone Lesions
Facial Monoplegia
No Emotional Movements
Bell's Phenomenon

- a. **Suprageniculate:**
  - Taste not affected, usually
  - Absence of stapedial reflex.
  - Impairment of salivary secretion.
  - Lacrimation affected.
  - VIIIth nerve may be involved.
- b. **Transgeniculate:**
  - Taste affected.
  - Stapedial reflex absent.
  - Impairment of salivary secretion.
  - Lacrimation affected.
- c. **Suprastapedial:**
  - Taste affected.
  - Stapedial reflex absent.
  - Impairment of salivary secretion.
  - Lacrimation not affected.
- d. ***Infrastapedial:***
  - Taste affected.
  - Stapedial reflex present.
  - Impairment of salivary secretion.
  - Lacrimation not affected.
- e. **Infrachordal:**
  - Taste not affected.
  - Stapedial reflex present.
  - Lacrimation not affected.

Extratemporal:

- Complete or partial facial paralysis, depending upon extent of nerve involvement.
- Associated evidence of injury or tumor.
- The chin moves in the midline when opening the mouth (branch for the posterior belly of the digastric muscle is not involved) (Tschiassny).

Cawthorne[21] places importance in localization in the sense of taste reaction. He feels that lacrimation testing is of doubtful value, although at times a lesion proximal to the geniculate ganglion can be defined when there is an interference to the secretion of tears on conjunctival stimulation. The most clear-cut demonstration of the importance of the tests of localization can be found in the case of tumor of the greater superficial petrosal nerve, reported by Tremble and Penfield.[27] In addition to the peripheral facial paralysis, tinnitus, and deafness, the sense of taste was impaired on the affected side, and there was a decrease in salivary secretion on the same side; also the greater superficial petrosal nerve was implicated as indicated by a quantitative decrease in lacrimal secretion after ammonia stimulation, as compared with the normal side.

ELECTRODIAGNOSTIC METHODS.

Recent advances in electrodiagnostic methods have given a most hopeful outlook in the management of facial nerve paralysis. The basic use of faradic and galvanic currents and study of the reaction of degeneration (R. D.) is still fundamental. The loss of the faradic response is considered important by Tickle, Sullivan, Cawthorne and others; Kettle, Lathrop and Martin do not think it a satisfactory test. The limitations of these qualitative tests should be realized. The R. D. does not appear during the first two weeks following an injury severe enough to result in nerve degeneration. There is no reaction of degeneration in supranuclear lesions. Lack of response to faradic stimulation is not in itself an indication for surgical intervention; many cases have recovered spontaneously in the face of a negative faradic test, because this test does not show whether regeneration is impending. A negative galvanic response is indicative of muscle atrophy and a contraindication for nerve surgery. A positive galvanic response has been known to be found even after ten and 20 years of paralysis and the nerve successfully grafted. The severity of the degree of the R. D. is directly proportional to the recovery time; the prognosis in a partial R. D. is an earlier recovery than in a full R. D. If a normal response is obtained, complete recovery can be anticipated. If a complete reaction of degeneration is found a guarded prognosis for complete recovery should be given. Only half the patients will have a satisfactory result. In spite of the limited value, serial testing of responses to galvanic and faradic stimulation is of practical importance.

The chronaxie method of electrodiagnosis is used in some medical centers. Chronaxie is the minimal time required for effective stimulation of a muscle nerve. Chronaxie measurements indicate the progress of regeneration and are of aid in prognosis.

The modern use of precise electrodiagnostic methods[28] have eliminated some of the uncertainty of the qualitative methods. This is due to the development of electronic instruments which deliver stimuli of measurable intensity and duration. The graphic analysis of the state of the nerve, such as in the strength duration curve, chronaxia tetanus ratio, and the rheobase ratio, furnishes information of the stages of degeneration, denervation and early signs of regeneration. Despite the shortcomings of the various electronic methods, including their technical intricacies and lack of early helpful information of nerve damage, they are a great step forward.

Electromyography, and audiographic methods of electrodiagnosis, is the most accurate procedure in predicting evidence of re-innervation. It is an expensive instrument requiring skilled and experienced technicians. Information of nerve regeneration can be obtained two to three months before voluntary movement can be observed. The appearance of the polyphasic nascent unit, the disappearance of fibrillation potential, and increase in the number of normal motor unit potentials signify signs of regeneration.

The appearance in electromyography of fibrillation action potentials is proof of nerve degeneration, appearing about three weeks after nerve injury. According to Taverner[29] fibrillation potentials can be detected by the seventh to the tenth day. He found in Bell's palsy that electromyography can predict accurately complete functional recovery if fibrillation activity of the facial muscles is not present. Denervation with incomplete recovery of function can be expected if fibrillation is present. According to Collier,[30] "a block to conduction can be diagnosed from true nerve degeneration by the absence of fibrillation potentials associated with reduced motor activity, while a mixed or partial degenerative lesion can be diagnosed by the presence of fibrillation action potentials in association with normal motor unit activity." Collier adds that even the most precise electrical methods cannot give us prompt evidence of degeneration; she feels that their chief value lies in repeated examinations to observe progress, but warns not to expect too much from a machine.

BELL'S PALSY.

Bell's palsy is regarded as a spontaneous unilateral peripheral facial paralysis arising in an isolated intrinsic lesion with no demonstrable evidence of other disease or injury. The current etiological concept is based on the ischemic theory in which the paralysis is thought to result from a sequence of events following segmental arteriolar spasm, with secondary ischemia, edema, and compression of the nerve in the bony facial canal. Many observers have noted the edema of the nerve in the early stage of Bell's palsy, and in cases of long standing, an atrophic fibrous strand. Kettel[31] feels that the pathogenesis is clear—an ischemia existing near the stylomastoid foramen; the nerve becomes unduly constricted, and then edematous. The swelling of the nerve in the bony canal results in compression of the adjacent blood and lymph vessels, giving rise to a vicious cycle. The process is reversible and may also affect the bone of the adjoining mastoid cells as well as the facial canal—an ischemic bony necrosis. Hall[32] confirmed Kettel's findings in three cases of Bell's palsy in which a decompression of the facial nerve was done. The nerves were highly edematous; yellow serous fluid was found in some mastoid cells, and the bone near the stylomastoid foramen was definitely soft.

The microscopic observations of the older pathologists demonstrated a parenchymatous peripheral neuritis, and degenerative changes of the axis cylinders and medullary sheaths in the peripheral part of the nerve were noted. The recent report by Jongkers[33] on the histological examination of the chorda tympani nerve in one case of Bell's palsy is interesting in this regard. He concluded that the process was an acute vascular disturbance. This was followed by an exudation of fluid, swelling of the nerve, and nerve degeneration; there was no evidence of inflammation. The question often asked is, "What causes the arteriolar spasm in the first place?" Hilger[34] and others have followed the explanation of older writers who consider Bell's palsy a dysfunction of the autonomic nervous system. Certain individuals have inherited an autonomic imbalance whose vasomotor response to stress situations result in segmental arteriolar spasm. The resultant ischemic paralysis may be mild and transient or may be severe and prolonged, producing an ischemic necrosis of the nerve.

As Hilger has stated, edema is not always found on exposure of the nerve. The cause of the paralysis is the ischemia of varying degree and the edematous compression of the nerve in the bony canal is a secondary factor. Collier[30] has stated that the site of the lesion is not always at the stylomastoid foramen, but Cawthorne[21] found 12 of 14 cases constricted at the stylomastoid foramen with swelling of the nerve immediately above it. His observations were made with the dissecting microscope. Kettel found that 20 per cent of his operated cases of Bell's palsy showed bony necrosis of the mastoid cells especially around the stylomastoid foramen. If the paralysis on recovery is followed by the crocodile tear phenomenon the nerve must be involved proximal to the geniculate ganglion and distal to the facial nerve nucleus.

The onset of the paralysis is sudden and is usually accompanied by pain about the ear. The pain is considered a vasodilating pain, and the intensity of the pain is regarded as an index of the severity of the ischemic involvement. The paralysis may be partial, complete, or even permanent. The process may be a transient conductive block affecting the myelin sheath, or may proceed to total nerve injury. The response to faradic response may be ever present, or may disappear 10 to 14 days after the onset of the paralysis. James and Russell[31] found that their cases took two courses: in 80 per cent recovery began in days or 2 to 3 weeks, and were completely recovered in 4 to 6 weeks. These were the cases of transient block with no evidence of nerve degeneration by electrical study. In the balance of the cases the muscles were paralyzed for two or more months, and recovery by regeneration took place in three to nine months. The electrical studies in these cases showed the reaction of degeneration. Failure to recover was rare, but the incidence of associated movements was seen in about half of this latter group.

Hyperacusis or phonophobia (Tschiassny), due to paralysis of the stapedius nerve, and loss of taste sensation due to paralysis of the chorda tympani nerve, are often found. Spontaneous recovery takes place in a matter of weeks or several months in 75 to 85 per cent of the cases. At present there is no precise way except perhaps electromyography of forecasting which cases will have some degree of residual paralysis or the sequelae of imperfect recovery. It is because of the high incidence of spontaneous recovery that many physicians adopt a hopeful attitude of wait and see, dooming some people to a life-long facial disfigurement. The answer at present seems to be an active program of medical management, with surgery reserved for those few cases whose study indicates exploration. The most important factor is maintaining the contractibility of the facial musculature until regeneration has been accomplished.

OTOGENOUS FACIAL PARALYSIS.

Facial paralysis originating from disease or injury in the temporal bone constitutes 10 to 15 per cent of the cases of facial paralysis. Two per cent of all cases of acute and chronic suppurative otitis media have a complicating facial palsy, .5 per cent belonging to the acute group. Less than 1 per cent of simple mastoidectomies are followed by facial paralysis. Kettel found postoperative facial paralysis seven times more common in cases of chronic otitis media than in cases of acute otitis media.

Facial paralysis associated with otitis media is uncommon. Decker[36] found 15 per cent of 400 cases of facial paralysis associated with otitis media—8 per cent acute and 7 per cent chronic. He advised exploration in those cases of chronic disease, and in those cases of acute otitis media in which the paralysis occurred 10 days after onset of the infection. Tschiassny[37] suggested daily examination with faradic current in these cases, and advised surgery if evidence of the reaction of degeneration developed. In acute otitis media recovery takes place as a rule; in chronic otitis media operation is most always indicated. Cawthorne[38] found that the destruction produced by chronic otitis media exposed the nerve in the posterior part of the tympanic segment, in the bend, or it may erode the bony external semicircular canal.

In facial paralysis accompanying acute otitis media, Fowler,[39] Tickle,[40] and Farrior[41] advise mastoidectomy if indicated. Decompression should be done if the faradic test is negative and if there is no evidence of returning function after two months. In chronic suppurative otitis media they advise an immediate radical mastoidectomy; if there is again no evidence of regeneration in two months a decompression operation should be done.

Riskaer[42] states that Lund found the incidence of facial palsy equal in chronic and acute infections, although Kettel found the incidence in chronic otitis three times greater. Riskaer investigated 100 cases of otogenic paralysis. In 53 cases the paralysis occurred preoperatively, 18 in acute and 35 in chronic otitis media. In the acute cases the prognosis for recovery was very favorable; in chronic otitis the prognosis was favorable. In 47 cases of postoperative paralysis, the 18 cases due to operative trauma presented a poor prognosis, while the 29 cases appearing after an interval had a favorable prognosis. He concluded that all patients with otogenic paralysis should be kept under observation (under adequate treatment for disease), except for the cases of immediate postoperative paralysis. Exploration should be done in the cases under observation if spontaneous recovery is not seen in one or a few months.

The cause of the paralysis in acute otitis media is considered to be of toxic origin. In chronic otitis media the facial palsy is associated with a cholesteatoma, or an osteitic process in the wall of the facial canal; therefore as Kettel[13] has observed, the cases of chronic otitis media with facial palsy, if treated in a conservative manner, will have a poor result in 50 per cent of the cases. He advised a radical operation and possible exploration of the nerve at the same time.

It is now generally accepted that the treatment of facial paralysis occurring in acute suppurative otitis media and mastoiditis is the appropriate treatment of the ear infection. Facial paralysis on the other hand, appearing in chronic middle disease, may require radical mastoidectomy and perhaps ration of the facial canal. In the acute case the progno- is better if the paralysis appears early in the course of disease.

The effect of the antibiotic era on otogenous facial paralysis has not been evaluated. In a case of acute otitis media recently treated by Dr. Cary Moon, a facial paralysis developed on the fourth day while the patient was being actively treated with antibiotics.

In the case of traumatic otogenic paralysis the indications for surgical intervention are well recognized. The indications for surgery in cases of facial palsy associated with temporal bone disease and fracture is presented separately. Lempert[14] considers the proper management of facial nerve injury is prompt recognition during operation, and immediate repair. If the nerve is compressed by inward fracture of the bony canal, the area about the injury is decompressed; if the nerve is cross-sectioned decompression is indicated; if a portion of the nerve is lost Lempert prefers rerouting and end-to-end anastomosis (produces natural emotional facial expression). If the operator is unaware of the accident, early exploration is safer in order to tell the nature of the lesion (edema, compression, severance). No reliance should be placed in the faradic test in this instance; the reaction may be positive for days even if the nerve is severed. Collier advised exploration in cases of otogenic paralysis, but recommends a three-week delay as favoring the process of regeneration. Tickle has advised prompt exploration in postoperative paralysis, but states that if the operator is doubtful that he has injured the nerve he can observe the daily reaction of the nerve to faradic stimulation and operate if the response is negative.

In the pre-Lempert days the nerve was regarded by many otologic surgeons with fear and trepidation. The use of the endaural and drill technique, improved illumination and magnification, has made the facial canal more a landmark than a structure to avoid as much as possible.

As pointed out by Sullivan[45] and others, the anatomical site of injury in temporal bone surgery is in order of frequency *1.* below and distal to the horizontal canal; *2.* the tympanic portion; *3.* vertical segment; *4.* pyramidal segment; *5.* after exit from stylomastoid foramen. Tickle found that the nerve is most frequently injured during simple mastoidectomy when the surgeon has gone too low in looking for the antrum; in radical mastoid surgery the injury is found most often on the floor of the antrum. Cawthorne[21] in 31 cases found 10 were injured in the tympanic segment, 14 at the pyramidal segment, and seven in the vertical segment.

Kettel[43] has noted that the prognosis in postoperative otogenous paralysis of the delayed type is favorable, and operation is necessary only when hemorrhage into the canal, or a progressive osteitis is suspected. The prognosis in the immediate type is grave and exploration imperative. In cases in which the continuity of the nerve has been destroyed at operation the outlook is a permanent paralysis; if the nerve is repaired at the site of injury the outlook is favorable in 90 per cent of cases.

If the case is not seen for some time after the original injury the problem, as in all types of facial palsy, requires surgical judgment, assessing all factors in each patient. Evaluation of the electro diagnostic tests will furnish important information of the status of the nerve and muscles, although Martin[46] and others think that these tests, other than electromyography, are of questionable value. Absence of the galvanic reaction and fibrillation on electromyographic examination is evidence of muscle fibrosis and contraindicate any nerve surgery. If evidence of impending nerve regeneration is at hand, operation should be delayed and physiotherapeutic measures instituted. Operation should be done if repeated electrical testing shows no sign of impending re-innervation.

Martin relies on clinical observation plus electromyography to decide on surgical intervention in peripheral traumatic paralysis. He places great importance in the presence or absence of muscle tone. If good muscle tone is present surgery can be delayed for six months; if lost, surgical intervention is necessary. He has found that the ultimate result is the same in spontaneous recovery as surgical repair; both have the same sequelae—contractures, tics, and associated movements.

Paralysis appearing 3 to 4 days after mastoidectomy is often caused by packing, compressing an exposed area of nerve; removal of the packing will result in early recovery. Slight traumatic edema, hemorrhage in the facial canal, or an unexposed osteitis may also produce a temporary paresis.

If the nerve is found to be inaccessible for repair, as in cases in which the gap involves the geniculate segment, hypoglossal-facial anastomosis is advised, often in conjunction with facial plastic procedures.

In extra temporal facial nerve injury, as Cody[1] has emphasized, nothing can be gained by "watchful waiting." Prompt surgery is indicated before the procedure is complicated by infection and scar formation.

FACIAL PARALYSIS FOLLOWING CRANIAL TRAUMA.

Facial paralysis is not an infrequent complication of basal skull fracture. It is said that the facial nerve is the second most common injured of the cranial nerves, exceeded only by the olfactory nerve. According to Grove[47] the facial nerve is usually spared in longitudinal fracture of the temporal bone, but is exceptionally vulnerable in transverse fracture of the petrous bone. In 90 per cent of cases the nerve injury is incomplete and temporary, and will recover with a complete return of function. Facial paralysis appearing one to two weeks after injury recover spontaneously in all cases.

Although some otologists believe in earlier decompression in selected cases, it is generally considered that most cases of facial paralysis following cranial trauma recover spontaneously, and exploration is limited to those cases which after careful observation, X-ray studies and electrodiagnostic testing offer some chance for success. In the cases of facial paralysis caused by a transverse fracture of the petrous pyramid, in which the injury is surgically inaccessible, hypoglossal-facial anastomosis is indicated. Lathrop[48] has found that if complete peripheral facial paralysis is present for over a year the prognosis for nerve surgery is almost hopeless. He has gained little help from X-ray studies, responses to the electrical tests, or other aids in localizing the lesion or managing the paralysis. He places greater importance on the history and physical examination. His success in handling these cases have been in proportion with the accuracy of the assessment of the individual case.

Kettel[49] has divided facial paralysis following temporal bone fracture into those that are immediate and those that are delayed. The delayed paralysis has a good prognosis and require conservative treatment. In a small number of the immediate cases, surgery is indicated; the indications are: *1.* prompt exploration if site of lesion is localizable and accessible; *2.* after two months of observation with no sign of returning function, and *3.* long standing cases if there is no atrophy of muscles.

In Turner's[40] series of 70 cases of facial palsy in closed head injuries, 34 were of the delayed type and 36 of the immediate type. All but two of the delayed type showed good recovery; in the immediate type three had no recovery, and six had an incomplete recovery with associated movements. He noted one case of bilateral traumatic palsy. He, too, poses the question —could early decompression hasten recovery and (or) prevent development of associated movements and facial contractures? If the cases recover slowly, Turner advises therapy to keep the facial muscles in good condition—a wire splint hook to prevent sagging, massage, galvanic stimulation, and practice of facial movements when recovery starts. In general the treatment of facial nerve injury after gunshot wounds is the same as post operative mastoid facial nerve injuries.

Kettel calls attention to an uncommon fracture restricted to the mastoid process and involving the descending segment of the nerve. He also noted that the prognosis in facial paralysis in head injuries is not so favorable as generally considered. He is an advocate of early exploration, if careful evaluation justifies the operation. In these cases, the estimation of the site of the lesion demands close otological and neurological study. Often the decompression must be postponed until the patient recovers from the effect of the cranial trauma. Surgically accessible usually means one peripheral to the geniculate ganglion, although Tickle, Tremble, Penfield and others have reported grafting in the lateral segment at or near the internal auditory meatus.

In recent years the trend is toward surgical intervention in selected cases, although, as illustrated by Turner's series of 70 cases, the number of cases is limited. In Cawthorne's[21] opinion, if signs of ear damage is found, especially if the middle ear is involved, operation should be considered.

Lathrop[51] has written on the aspects of facial nerve injury in warfare. He noted the multiple wounds and accessory contusions and fracture in battle casualties. Facial nerve injuries in this regard involve loss of nerve continuity and need for nerve grafting and rerouting. He appeals for constructive efforts to avoid facial deformity. Regarding the incidence in war, Cawthorne[21] recorded six cases of facial nerve injury, chiefly at the exit, in 2,277 air-raid and battle casualties. It has been reported that facial nerve injuries occurred in 46 cases of 513 peripheral nerve war injuries or 3.6 per cent, and in 21 cases in 53 cranial nerve war injuries or 40 per cent.

CONGENITAL FACIAL PARALYSIS.

Facial paralysis in infancy can be caused by injury to the nerve by obstetric forceps, or by pressure on the face by the bony prominence during birth. Congenital facial diplegia, or Mobius' syndrome, is a rare condition due to nuclear agenesis.[52] It is associated with other cranial nerve palsies. Intracranial trauma and subarachnoid hemorrhage with extension of bleeding into the temporal bone have been reported to affect the facial nerve. Congenital facial paralysis may be associated with other congenital defects, syndactylism, club feet and absence of muscle groups.

FACIAL PARALYSIS ASSOCIATED WITH POLIOMYELITIS.

Facial paralysis associated with poliomyelitis has been more evident in recent years, no doubt due to the increased number of cases of poliomyelitis. In an analysis of the incidence of involvement of the cranial nerve in 500 cases of poliomyelitis, Engler and Missal[53] found that the facial nerve nuclei were involved alone in 55 cases, and in combination with other cranial nerves[2,5,6,9,10,11,12] in 45 cases. Next to the nucleus ambiguus group (202 cases) the facial nerve nuclei are most commonly involved.

FACIAL PARALYSIS IN THE SYSTEMIC DISEASE.

Facial paralysis is associated with central nervous system disease (encephalitis, progressive bulbar palsy), with infectious diseases (diphtheria, meningitis) and with toxic substances (alcohol, arsenic).

Facial palsy is common in the Guillain-Barré Syndrome, occurring in half of the cases and in 85 per cent of these bilateral.

Facial palsy may also result from cell infiltration in leukemia, and is seen in infectious mononucleosis.[54]

Facial diplegia may be caused by many types of lesions, being associated with bilateral otitis media, infectious diseases, basal skull fractures, alcoholic polyneuritis, meningitis and in leprosy and malaria.

FACIAL PARALYSIS ASSOCIATED WITH THE UVEO-PAROTID SYNDROME.

Uveo-parotid fever is now considered a form of sarcoidosis. It is an uncommon disease, affecting younger individuals, and characterized by constitution symptoms and painless, firm swelling of the parotid glands. The ocular involvement, while it is usually a uveitis, may involve any tunic of the eye. Paralysis of the facial nerve, unilateral or bilateral, is seen in over 4 per cent of the cases. The prognosis is favorable; the paralysis appears and subsides with the parotid swelling.

HEMIFACIAL SPASM.[55]

Paroxysmal facial spasm, limited to all or a portion of the muscle supplied by the facial nerve, is a disease of unknown cause. It is characterized by irregular, intermittent contractions of the facial muscles, usually unilateral but sometimes bilateral. The contractions may vary from small twitching of the orbicularis oculi to severe violent spasm of all the muscles on one side of the face. Facial spasm is a disease of adult life, involuntary, occurring at times during sleep and aggravated by stressful situations. It is to be differentiated from facial tic, a habit spasm or compulsion neurosis, in which the muscles involved are supplied by several nerves.

The cause of primary hemifacial spasm lies in the facial nerve, although the exact nature is undetermined. Electromyographic studies have resulted in patterns suggestive of an irritative lesion of the nerve trunk.[56] Secondary hemifacial spasm is a development following intracranial lesions or is one of the facial anomalies following regenerated facial paralysis.

Facial spasm following recovery from paralysis has been considered to be of central origin, but evidence seems to favor a lesion of the lower motor neuron.

The treatment is difficult; medical management has been of little value, and surgical treatment has been occasionally successful. Decompression of the facial nerve, or neurolysis, has relieved some cases, but usually, as in a case recently observed by us, the spasm returns after relief for a year or less. Cross anastomosis, section of individual branches of the nerve or alcohol injected into the nerve (by the O'Brien technique as recently described by Greer[57]) are procedures not too satisfactory.

Scoville's operation, partial section of the facial nerve trunk just distal to the stylomastoid foramen, is based on German's technique of partial sectioning of three branches of the facial nerve distal to the parotid gland. Scoville's[59] results appear to be excellent.

Lewis[60] recently reported on three cases of hemifacial spasm in which he performed endaural decompression and neurolysis of the facial nerve. His results were temporary improvement, but then return of spasm in three cases, and complete relief for over a year in the third case. He next proposes to follow the decompression with a modified Scoville's procedure if the spasm is not relieved by the decompression alone.

In Williams'[56] and his co-workers' series of cases the most interesting result following decompression of the facial nerve for hemifacial spasm was the disappearance of the synkinesis, or associated movements of the facial muscles. This suggested to them the hypothesis of "cross-firing", or the excitation of fibers by impulses traveling over adjacent fibers, rather than the concept of branching or misdirection of nerve fibers during regeneration to account for the associated movements. Their observation of a definite thickening of sheath of the nerve in the facial canal suggested that the etiology of facial spasm is a compression of the nerve and its blood supply. Although their patient achieved a measure of relief following neurolysis there was a tendency toward recurrence of the spasm.

SYNDROME OF THE CROCODILE TEARS.[61]

The syndrome of the crocodile tears or unilateral lacrimation on mastication after recovery from facial paralysis has been confused with the epiphora that results from paralysis of the lower lid in facial palsy. The cause is said to be a deviation of the regenerating axones from their normal course. The syndrome is part of the abnormal facial movement following recovery of facial nerve injury, such as closing the eye automatically draws up the corner of the mouth. Fowler[62] showed experimentally that the associated tic-like movements are caused by splitting of the axones in the neuroma that form at the site of the injury. The regenerating fibers do not always follow the same path as before injury. The facial nerve injury in this instance is proximal to the geniculate ganglion. During regeneration some secretory motor fibers which formerly ran with the chorda tympani pathway became misdirected in the greater superficial petrosal pathway to the lacrimal gland. Section of the greater superficial petrosal nerve has relieved the symptoms.[63]

This syndrome is analogous to the auriculotemporal syndrome, a sequel to suppurative parotitis, parotid surgery and wounds of the face. The misdirected regenerated parasympathetic salivary fibers produce a situation of facial flushing and sweating over the parotid area when the patient eats. The symptoms have been relieved by intracranial division of the glossopharyngeal nerve.[64]

MEDICAL TREATMENT FOR FACIAL PARALYSIS.

The current trend in the medical management of Bell's palsy is based on the hypothesis of ischemic injury to the facial nerve. The early use of the vasodilating drugs, as emphasized by Hilger,[34] has brought interest and hope in preventing the inevitable consequences of nerve degeneration. If by medical means the sequence of arteriolar spasm, capillary dilation, edema, and compression of the nerve can be retarded or stopped during the stage of physiologic block, early recovery and an increase in the number of spontaneous restorations of function can be expected. If prompt and vigorous treatment fails to produce signs of recovery, careful evaluation of the severe case may suggest earlier surgical intervention before the condition has advanced to the stage of nerve degeneration.

The vasodilating drugs used at the present time are those which act directly on the blood vessels, such as histamine and nicotinic acid; and the measures which relieve vasospasm and

increase blood flow, sympatholyti
blocking agents and stellate gar
others have reported on the rapid
palsy with intravenous histamine
19 cases of facial paralysis witl
dose" method. He termed the ra
nomenal. Articles by Swan[67] ar
that homolateral stellate gangl
block can effectively cure early ca

The successful treatment of ear
Cortisone, as reported by Robisor
again raised the question of how
of treatment in a disease which h
spontaneous recovery. It has be
that in cases treated with Corti
recovery has been very prompt, n
weeks. Rothendler[20] states that th
edema and inflammatory reaction
has suggested that the mechanism
not necessarily at the site of the
the fundamental factors which br
jury. Taverner[71] found in a cont
the incidence of denervation of
palsy, that Cortisone influenced
nervation nor hastened the recov
nervation.

The use of intravenous procair
paralysis is designed to break u
and accelerate vascular flow. Fo
100 mg. of niacin four times dail
250 cc. of a .01 per cent procai
venously twice daily.

There is little doubt that the

tressed by his disfiguring malady. Indeed, the psychologic rehabilitation of the patient and adjustment to his facial deformity, as stressed by Lathrop, is of extreme importance.

A great contribution of physical therapy has been the prevention of contractures and deformity of denervated facial muscles. The muscle atrophy and early fibrosis, so apparent in the face, is due to the anatomy of the facial muscles; they are inserted directly into the skin or subcutaneous tissue without intervening subcutaneous fascia and are devoid of muscle fascia surrounding the individual muscles. The over stretching of the paralyzed muscles by the force of gravity and the pull of the muscles on the sound side can be helped by supporting the drooping mouth. Various methods have been advised; an intraoral splint, adhesive support, a wire prop or splint hooked around the ear, and the use of a simple cigar or cigarette holder held in the corner of the mouth. It can be added that the fascial strip operation has been performed to hold up the muscles until the nerve regeneration restored muscle function.

The role of galvanic stimulation of the facial muscles has been controversial. Authorities now generally agree that it is a valuable adjunct, and electromyography has shown that muscle contractibility is helped by daily interrupted galvanism. Osborne[73] advised a modulated alternating current with a carrier frequency which is found by making an intensity frequency curve. Rodriguez and Skolnik[28] have stated that physical measures are used to retard or prevent muscle fiber degeneration and electrophysiologic and chemical changes. They recommend a sound program of electric stimulation.

Massage, properly performed, has been advised to stimulate venous and lymphatic circulation and improve muscle tone. At the first sign of return of active movement, exercise under proper guidance is urged, and Cawthorne[74] has written of the use of a tin whistle or mouth harp in this regard. The re-education of the facial muscles by mirror study can be a rewarding exercise. A placid and relaxed facial expression should be cultivated (Spiller).

It has been stated that the medical management can be governed by the result of serial electrical testing. If normal electrical excitability is present 10 days after the onset of the paralysis, recovery can be expected in 3 to 6 weeks in the average case, and therapeutic measures need not be too active. If, on the other hand, the patient presents signs of a progressive reaction of degeneration, the course of recovery will be prolonged and persistent therapy is necessary. In this case the patient should be told that some defect in returning function will likely result. Even under the most expert care, the facial muscles which recover after denervation will tend to develop contracture and associated movements.

Park and Watkins[22,23] feel that physical medicine is important more to relieve anxiety and prevent emotional disturbances. They think that therapy does not significantly alter the course of the disease, although functional improvement can be achieved by muscle re-education. Others also feel that no form of treatment has any influence on the outcome, and the final result depends on the severity of the initial lesion.

SURGICAL TREATMENT.

Although preceded by a number of surgeons the technique of intratemporal repair of facial nerve injuries and the development of the autoplastic nerve grafts is based on the early work of Bunnell[75] and on the brilliant investigations of Ballance and Duel[76] in the early 1930's. Since that time considerable progress has been made in solving some of the problems involved. Continued differences of opinion exist relative to the indications for and the value of the surgical management of facial paralysis. The indications for surgery in cases of facial palsy associated with temporal bone disease have been considered previously.

It is in the surgical management of Bell's palsy that great divergence of opinion exists. James and Russell,[35] for instance, condemn the early decompression operation for Bell's palsy based on no recovery in 6 to 8 weeks and a negative faradic response. They feel that surgical treatment can hope to be successful only if done very shortly after the onset of the palsy. They and others conclude that if a transient block of conduction is present, recovery is assured and surgery is of no help. If the nerve degenerates the regenerative process will have to proceed in any event. The question is, does decompression retard degeneration, hasten regeneration or avoid sequelae? Many feel there is no difference between the result of spontaneous regeneration and surgically assisted regeneration. There is no method of determining whether the degenerated nerve is totally destroyed or whether the process involves only the axone with preservation of the supporting structures (Schwann cells and neurolemma tubes). In the latter instance spontaneous recovery with probable sequelae can be expected; therefore, the prevention of the secondary edematous compression of the nerve in its rigid canal should be a goal to be sought, because it is this injury that produces the complete degenerative lesion. The primary ischemia affecting the myelin sheath is of temporary nature, and this stage of physiological block will go on to complete recovery. Regarding early operation Kettel,[77] however, found that even if decompression was performed a few days after the onset of the paralysis there was no guaranty that full mobility would be recovered. He concluded that the most severe cases had the most profound primary ischemia, and the results depend upon the degree and duration of the dysregulation of the vasa vasorum.

Collier[30] states that there is no electrical test available that will foretell which nerve will likely progress to degeneration. She contends that if the clinical evidence of severe nerve damage is used as an indication for surgery, many patients will be subjected to operation who would recover spontaneously. To prevent degeneration, the time to operate is during the stage of physiological block, before the faradic response is negative, and a large number of these patients will recover anyway. Collier states that an ischemic block can last for six weeks or longer; therefore, the use of an arbitrary time limit, or two months as an indication for surgical intervention, is not reliable. She made a plea for continued study of the role of decompression in Bell's palsy in centers with adequate research facilities.

The advocates of early decompression in Bell's palsy cite evidence to the effect that relief of pressure on an edematous nerve enclosed in a bony canal can be most effective in the early stages of the disease. Sullivan[45] emphasizes that the early effects of the "dysregulation of the circulation" is confined to the myelin sheath; continued pressure affecting the Schwann cells and axis cylinders produce a complete degenerative lesion. Early relief of this pressure is necessary, and the nerve will proceed to rapid recovery. If the nerve is observed six months or more after unrelieved pressure all that remains is a fibrous strand.

Collier[30] quotes the experimental work of Denny, Brown and Brenner[78] on the effect of pressure on a nerve after occlusion of the blood vessels. The temporary block of nerve conduction (due to ischemia affecting the myelin sheath) is changed by the sequence of vascular stasis, increasing edema and nerve compression, to a degenerative lesion damaging the axis cylinders and the Schwann cells. This work would appear to favor the proponents of early decompression and establish the ischemic theory of Bell's palsy.

According to Kettel[31] the operation should be considered in *1.* those cases in which signs of beginning mobility have not appeared after an observation period of two months; *2.* in those cases in which spontaneous recovery of mobility has ceased before recovery has been obtained; *3.* in those cases exhibiting relapsing paresis. The two-month waiting period allowed *is based on experience: Kettel found that all cases of* otogenous paralysis, which recovered spontaneously, showed signs of recovery in two months; those that did not, showed no signs of recovery when the two-month period had elapsed. Kettel has found that the Faradic response is valuable, but not absolutely reliable. He has agreed that there has been no general agreement on the indications for surgery.

Cawthorne,[38] in his operated cases, found edema of nerve with a constriction at the level of the stylomastoid foramen. In cases of long standing paralysis the nerve was reduced to "a shrunken and reddened strand." It is Cawthorne's practice, first to assess the severity of the case and judge the prospect of spontaneous recovery. If the paralysis is complete and severe, and there is no response to faradic stimulation within a month there is a probability of permanent residual paresis, and operation is advisable. The decompression insures the maximum amount of recovery in his hands, with less likelihood of the defects that follow regeneration. If on exploration a fibrous strand is found the nerve should be grafted. Cawthorne believes the electrical reactions helpful, although for adequate testing an anesthetic may be necessary in children. The R. D. may appear as early as 72 hours, or as late as 14 days. He states that the "present opinion favors exploration of the nerve trunk in cases of continued paralysis where the lesion is thought to be surgically accessible."

The factor of severe and continued pain and loss of taste sensation, in assessing the severity of the lesion, is used by some as an indication for surgery.

The case of Bell's palsy seen several months after the onset requires careful study and evaluation. The role of electromyography in this regard is most important.

Tickle bases his indications for decompression in Bell's palsy on the responses to electrical stimulation and the tone of the facial muscles, especially the angle of the mouth in response. If the faradic test is negative, the angle of the mouth drooping, and the patient has shown no signs of improvement in two months, surgery is indicated. In general his indications for surgery are the same as Kettel's.

Martin[79] feels that medical therapy and physiotherapy should be tried first in Bell's palsy. If after two months there is no return of function or there is a relapsing paresis, surgery is indicated if a carefully taken history and examination (he does not think electro diagnostic tests are entirely reliable) confirm the surgical decision.

Regarding the end result of surgical intervention Kettel[80] has reported on 69 cases in which nerve grafting and nerve suture was done for a surgical lesion of the facial nerve. He states that even the most successful operation will not be able to restore facial function completely. In the most favorable group, 90 per cent clinically satisfactory result was obtained; where the muscles had degenerated partially or when technical difficulties were encountered, as in extratemporal repair in a bed of scar tissue, the operation had to be regarded as an experiment. In other articles Kettel[31] has recorded his results in Bell's palsy and otogenous paresis. In the latter group he did 50 decompression operations. All the patients recovered partial or complete mobility. In 50 cases nerve grafting or nerve suture was performed. Ninety per cent of these cases recovered, even in the face of extreme technical difficulty or poor facial musculature. Kettel's results in the Bell's palsy cases are divided according to his three indications for operation. In the first category (no recovery in two months) only 10 per cent recovered complete mobility. The remaining cases resulted in improved appearance and partial facial movement. All cases had synkinesis, slight to severe. In the second category nine out of 12 were improved, three had no effect, and one showed an immediate disappearance of a contracture (release of reflex nerve irritation). In the third category (relapsing cases) the mobility improved in all eight cases, and two recovered completely.

As Martin has remarked, it is difficult to find in the literature the percentage of surgical failures. In an abstract of an article by Kettel[81] on the surgical aspects of Bell's palsy no mention was made of the final result. Kettel operated on 269 patients with peripheral facial palsy; 97 belonged to the Bell's palsy group. In three out of 97 the nerve was edematous; in three atrophic; in 17 patients an ischemic necrosis with exudation was found in the mastoid cells; in 20 cases the facial canal wall was soft; microscopically aseptic bony necrosis was demonstrated.

Facial nerve surgery requires careful, meticulous technique, with the aid of all the facilities of modern temporal bone surgery. The choice of facial nerve repair, whether it is a simple decompression; a decompression and end-to-end anastomosis; decompression, rerouting and end-to-end anastomosis; or decompression and nerve grafting, depends on the nature of the case and the experience of the operator.

The methods of exposure of the canal and the technical details of the nerve surgery will not be considered here.

ANASTOMOSIS.

If the central end of the facial nerve is not accessible for nerve grafting, as in tumors of the acoustic nerve, anastomosis of the facial and hypoglossal nerve can be accomplished. Hypoglossofacial anastomosis relieves the asymmetry of the face in repose, and there is an improvement in voluntary facial movement, especially about the mouth, but rarely the forehead. The result is inferior to that following intratemporal repair. The reactions of the facial muscles to emotional expression will be absent. Facial-hypoglossal anastomosis is preferred to use of the spinal accessory nerve anastomosis because of the associated movements and shoulder deformity; the hemiatrophy of the tongue after section of the hypoglossal nerve is not of importance. Love and Cannon[82] prefer spinofacial nerve anastomosis. Often the restoration can be improved by combining anastomosis with fascial slings.

PLASTIC PROCEDURES.

The correction of facial distortion by mechanical suspension of the paralyzed muscles is useful not only when the muscles are fibrosed and atrophied, but also as an adjunct to nerve surgery, or even as temporary support of the sagging facial muscles when ultimate regeneration of the nerve is anticipated. The subcutaneous insertion of loops of fascia lata,[83] tantalum wire and other materials by various techniques, has been successful in many instances in restoring facial symmetry. The use of muscle transplants has also been advocated, and combined with fascia lata support. Owens[84] has written on the implantation of fascial strips through the masseter muscle, and the transplantation of muscle strips from the masseter and temporal muscles.

SEQUELAE OF FACIAL PARALYSIS.

The greatest problem in facial paralysis is how to avoid or lessen the sequelae of an unregenerated or poorly regenerated facial nerve. How can the facial symmetry, the full voluntary and emotional movements present before the paralysis, be restored? How can the spasm, contractures, associated movement or synkenesis and deformity be prevented? There is evidence that progress is being made in means of recognizing and preventing nerve degeneration and muscle changes, and in the treatment of the residual disturbances. A great obstacle has been the hopeful and procrastinating attitude of many physicians in the anticipation of possible spontaneous recovery, and disregard of the medical and physiotherapeutic possibilities in the management of facial paralysis. The thinking now is in terms of avoiding nerve degeneration and the inevitable defects in function following nerve regeneration. James and Russell plead for some form of therapy which will avoid Wallerian degeneration and its consequences. In studying 58 cases of Bell's palsy they noted 30 cases of complete recovery and 28 cases of incomplete recovery. In 13 of the latter cases the cosmetic result was unsatisfactory. This included associated and mass movements causing overuse of certain muscles—the patient smiles and winks at the same time.

The cause of the sequelae is thought to be a misdirection of the regenerating axones, or a splitting of the axones at the site of the injury. In the process of regeneration after degeneration the axones often do not follow the same path as before the injury. The theory of cross-excitation of fibers at the site of the lesion and the theory of changes in the nuclear excitability leading to synchronous firing has also been proposed. The cause of the muscle deformity lies in the changes in the delicate facial musculature which, unlike skeletal muscles, are inserted directly into the skin and subcutaneous tissue, and have no definite muscle fascia surrounding the individual facial muscles. The over-stretching of the denervated muscles by the force of gravity and by the muscular pull of the opposite side of the face accelerates the muscle atrophy and fibrosis.

Taverner[29] found that the sequelae can be predicted; they developed only in those patients who demonstrated fibrillation on electromyographic examination. The residual dysfunctions in his cases included contractures, associated movements, spontaneous twitchings and paroxysmal lacrimation when eating. Electromyographic study has also shown that physiotherapy especially galvanism retards atrophy and fibrosis.

TUMORS.

Tumors arising from the facial nerve are rare. According to O'Keefe[35] only 15 cases have been recorded in the literature. Neurinomas of the facial nerve are characterized by early progressive facial paralysis and evidence of temporal bone involvement. It is a benign tumor arising from the cells of the sheath of Schwann and grows by expansion. The site of origin is usually the vertical portion of the nerve; occasionally th horizontal portion[86] is the initial site, and sometimes the locall destructive and expansive growth is first seen after destruc tion of the bony labyrinth.

Bogdasarian[87] states that Altmann first pointed out the clin ical features of neurinomas of the facial nerve: *1.* youn adults; *2.* facial paralysis developing gradually, sometime suddenly, occasionally relapsing; *3.* mass in external auditor canal; *4.* pain; *5.* chronic purulent otitis media complicating *6.* widespread destruction of the temporal bone.

The diagnosis is confirmed by biopsy of the tumor mass i the external auditory canal. It is not possible to diagnose tumor of the facial nerve if the only sign is a Bell's palsy, except a strong index of suspicion should be present if continuous pain is found, or if the paralysis develops gradually Roentgenologic examination may or may not be helpful.

The prognosis is favorable except for nerve function. Radiation therapy is not effective. The treatment is removal o the tumor and the restoration of facial movement by method indicated by the individual case. In Collier's and Thomson' case the neurofibroma involved the vertical portion of th nerve. The tumor was excised and a nerve graft inserted. There was a gradual return to facial function.

Tremble and Penfield reported a case of perineural fibroblastoma (neurinoma) of the greater superficial petrosal nerve discovered in the course of exploration of the facial canal. Love[88] has described a case of intrapetrous neurofibroma, originating from the VIIIth nerve within the petrous bone. The patient presented a unilateral deafness and a progressive facial palsy. The palsy was later successfully managed by a spinofacial anastomosis.

In the case of acoustic neurinomas, the presence of a facial paralysis is a late sign and appears last in the sequence of *1.* acoustic symptoms; *2.* trigeminal and cerebellar symptoms; *3.* headaches and evidence of increased intracranial pressure. Close observation of the orbicularis oculi muscle may detect early facial nerve involvement. In the case of inflammatory cerebello-pontine angle lesions, the facial paralysis appears early. As pointed out by Kennedy[89] inflammatory lesions have a direct pressure effect on the facial nerve, whereas a tumor has a stretching effect. Inasmuch as the sensory portion of the nerve is affected by lesions in this locality proximal to their cell bodies, and the motor fibers distal to the motor nucleus, inflammatory processes tended to spare the sensory fibers, while in the case of new growth the sensory changes appeared earlier.

Miller and Uihlein[90] have written of progressive and intermittent facial paralysis in patients with tumors of the petrous ridge. Intermittent facial palsy is uncommon, but was present in their case of epidermoid tumor of the petrous ridge. The various types of tumors of the petrous ridge usually exhibit a progressive facial paralysis.

In 1950 Kettel[91] reported a very rare case of malignant intratemporal sarcoma of the facial nerve. The tumor was found on decompression of the facial nerve for Bell's palsy. The patient did not present anything abnormal on otologic or neurologic examination. The tumor was a neurogenous spindle cell sarcoma of mesodermal origin. The tumor was resected and the defect nerve grafted. The second case of neurofibrosarcoma of the facial nerve was reported by Guttman and Simon[92] in 1951. These tumors are relatively malignant and, as with neurinomas, usually involve the descending portion of the nerve and are characterized by a slowly progressing facial paralysis. They are differentiated from facial paralysis in carcinoma of the middle ear and mastoid, by the history; the paralysis in carcinoma appears last after pain, deafness and otorrhea.

In addition to carcinoma of the middle ear the facial nerve can be paralyzed by pressure produced by intratemporal epidermoids and glomus jugulare tumors. Jefferson and Smalley[93] reported six cases of progressive facial palsy due to painless latent non-infected petro-mastoid epidermoids. These cases are sometimes considered an unusual type of Bell's palsy, but the slow development of the palsy, X-ray study by careful technique, absence of otitic suppuration, and finally exploration will establish the diagnosis. This tumor is probably of congenital origin and affects the facial nerve solely by pressure.

MELKERSSON'S SYNDROME.

In 1928 Melkersson[94] described an unusual syndrome consisting of *1.* recurrent peripheral facial paralysis, *2.* chronic angioneurotic facial edema, *3.* lingua plicata. The exact cause of the syndrome is unknown; it is thought to be a vague disorder of the vasomotor system and allied to our present concepts of the etiology of Bell's palsy. Recovery of the facial paralysis is slow but is usually complete, without residual facial distortion. In the case reported by one of us, the recovery following facial plastic surgery has been complete, and she has had no further attacks of paralysis.

The treatment[95,96] of Melkersson's syndrome has been twofold: *1.* plastic correction of the permanent areas of the facial edema; 2. the medical and surgical management of Bell's palsy, as indicated in each individual case.

TIC DOULOUREUX OF THE NERVUS INTERMEDIUS.[97]

Tic douloureux of the nervus intermedius, sometimes called idiopathic geniculate neuralgia, is a rare but definite entity. It is characterized by severe tic-like paroxysm of pain deep in the affected ear with a trigger area in the external canal. In recent years it has been shown by Furlow and others that the disorder originates in the nervus intermedius and can be relieved by section of that nerve. The condition should be differentiated from herpes oticus, trigeminal neuralgia and glossopharyngeal neuralgia. Section of the nerve results in ageusia of the anterior two-thirds of the tongue, reduction in salivary secretion, and hypesthesia of the tympanic membrane and adjacent part of the external auditory meatus.

HERPES ZOSTER OF THE GENICULATE GANGLION.

The concept of a herpic involvement of the geniculate ganglion or herpes oticus, producing otalgic and herpetic lesion in the external ear, was part of the monumental work of Ramsay Hunt[98] on herpes zoster of the cranial nerves a half century ago. Hunt visualized a posterior poliomyelitis of the sensory portion of the facial nerve, involving the ganglion alone or extending to involve the facial and auditory nerves—herpes oticus, herpes oticus with facial palsy, herpes oticus with facial palsy and auditory symptoms, and herpes oticus with facial palsy and Meniere's symptom complex. The disease is characterized by history of pain in the affected ear, preceded by symptoms of malaise and fever and followed by a vesicular eruption in the geniculate zoster zone of the auricle (see Fig. 8).

The cause of the facial paralysis in Hunt's syndrome is said to be either direct extension of the herpetic inflammation to the nerve trunk or pressure of the inflamed ganglion on the nerve. A conception of the site and extent of the facial nerve lesion can be made by using the topognostic analysis of Tschiassny, which has been mentioned previously. The prognosis for complete recovery of the paralysis is favorable.

It is also well to recall that paralysis of the facial nerve may accompany herpes zoster of the other sensory cranial nerves, especially the Vth and Xth. The exact nature of this simultaneous involvement is unknown, but is allied to the intricate overlapping anatomical network of the cranial nerves.

It is interesting to note that the complication of facial paralysis following a myringitis bullosa is regarded as a virus infection and related to Hunt's syndrome.

GENICULATE NEURALGIA.

Allied to the symptomatology of herpes zoster of the geniculate ganglion, but without the zoster herpetic vesicles, is a clinical entity at times diagnosed as geniculate neuralgia or neuralgia of the sensory portion of the facial nerve. The etiology is uncertain but is presumed to be of virus origin. The condition is self limited and characterized by a unilateral dull aching deep in the ear, with extension of aching to the cheek and forehead. Often there occurs an extension of the process to the VIIIth nerve producing mild vestibular and cochlear symptoms.

SURGICAL REPAIR OF THE PERIPHERAL FACIAL NERVE.

The integrity of the peripheral facial nerve may be interrupted, from trauma, as a complication of surgery upon the various tissues of the face, secondary to infiltration by a neighboring neoplasm, rarely by a primary tumor of the nerve, and probably still more rarely, but conceivably, by an adjacent inflammatory process. The resulting facial paralysis may be partial or complete, depending upon the site of the injury and the branches involved.

Bunnell,[99] Maxwell,[100] McKenzie and Alexander,[101] Lewis,[102] Ross,[103] Alexander and Davis,[104] Conley,[105] and others have recorded their experiences and opinions regarding the management of peripheral facial nerve injuries.

The best time for repair of the defect in the facial nerve, in the case of trauma, is when the lacerations are repaired. In the event of the necessity for resection of the nerve, as in the instance of neoplasm, the repair should be accomplished at this time of initial surgery. If for some reason immediate repair cannot be accomplished, then one should attempt to mark the severed ends as an aid to future recognition. The use of the faradic stimulator and magnification will be found to be most helpful in locating the various nerve segments. Theoretically, the time interval between the injury and attempted repair of the nerve should be infinite, providing the receptive muscle fibers are contractile, as evidenced by response to galvanic stimulation. Once atrophy and fibrosis have developed, secondary to lack of nerve stimulation, to the degree that contraction of the fibers is impossible. then the possibilities must be considered hopeless as far as rehabilitation of the facial deformity by reinnervation is concerned. The earlier the attempt at repair the better results one may expect.

In the repair of the nerve direct, end-to-end anastomosis is the procedure of choice. The nerve segments should be mobilized so that no tension exists, and the ends, freshly trimmed if necessary, are carefully approximated and loosely immobilized by 8-0 atraumatic silk sutures. The sutures should be preferably placed only in the nerve sheath, but when dealing with the smaller radicles, according to the technique devised by Conley,[105] such a procedure is impractical, and a through-and-through suture is utilized. Good viable surrounding tissue, such as muscle, will increase the chances for a good result.

When the gap in the nerve defect is too great for primary approximation, even after mobilization and rerouting, then a free graft is used. It apparently makes little difference whether the graft is a motor or sensory one, whether it is reversed, or whether it is fresh or partially degenerated; for, according to Ross, the graft functions only as a channel through which regenerated fibers travel to gain access to the distal or peripheral segment. From six to 12 months following injury is the wide variation of time stated as the limit for anticipating satisfactory results. Caldwell[106] has reported a remarkable case in which three 6-cm. grafts were sutured to the proximal segment, with the distal end of the graft not attached to the nerve but implanted in facial muscle, with good resulting function.

The donor nerve of choice in cases of facial nerve graft would seem to be the great auricular, in view of its accessibility, size in caliber and length, and its branches. The latter anatomical fact is advantageous when the necessity for a multiple branch is present. Other donor nerves that have been and may be used for grafts are those of the thigh.

If the facial nerve is injured near the stylomastoid foramen and primary anastomosis or free grafting is not possible, even after mobilization and rerouting, then anastomosis of the proximal facial with another cranial nerve, such as the hypoglossal, spinal accessory or glossopharyngeal, may be performed. The facio-hypoglossal anastomosis is preferred by most surgeons.

Although the above-mentioned procedures are successful in alleviating the facial deformity, as in intratemporal repair, the patient must be informed of the inadequacies in the results. These are: the varying degrees of mass motion, lack of restoration of emotional responses, hemifacial spasm, and associated involuntary muscle movements of the formerly paralyzed groups in the case of anastomosis with another cranial nerve.

In order to improve the results of peripheral facial nerve repair, supplementary surgical procedures, such as the insertion of fascial and wire supporting slings, the transposition of muscle, and face lift, may be performed with benefit. A program of re-education in regard to facial movement, by proper practices and exercises, will be useful in obtaining the maximum results. Various types of temporary facial supports are advised during the convalescent period while waiting for recovery, which may be a matter of from six to 12 months. In recovering, improvement in facial muscle tone is first noted, to be followed by voluntary movement, and lastly the exhibition of some more or less response of the emotional type.

CONCLUSION.

An anatomic-clinical study of the facial nerve and its diseases is presented, together with a survey of some of the viewpoints on the management of facial nerve paralysis. Many interesting considerations have been omitted; there is a need for a comprehensive treatise on this subject.

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OTOLARYNGOLOGICAL POSTGRADUATE COURSE.

Saint Luke's Hospital and the Cleveland Otolaryngological Society will present a postgraduate course May 9, 1956, in Prentiss Auditorium, Saint Luke's Hospital of Cleveland. 11311 Shaker Boulevard, Cleveland 4, Ohio.

Guest speakers will be Alden H. Miller, M.D., Associate Clinical Professor of Otolaryngology, University of Southern California, Los Angeles, Cal., and Kenneth M. Day, M.D., Professor of Otolaryngology, University of Pittsburgh, Pittsburgh, Pa. Other participants in the program are H. C. Rosenberger, M.D., Fred W. Dixon, M.D., and William F. Hulse, M.D.

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NECK DISSECTION: ITS ROLE IN CANCER OF THE LARNYX.[*][†]

GEORGE F. REED, M.D.[*] (By Invitation),

Boston, Mass.

In the treatment of cancer of the larynx, we are especially fortunate in having the cervical lymphatic area as a barrier to more distant spread of metastasis. Not only are the cervical lymph nodes very effective "cancer stoppers," but the area lends itself very nicely to *en bloc* resection, both by itself and in continuity with resection of the larynx.

Are we taking full advantage of this natural barrier, however? The answer to this question lies in a statistical evaluation of cases previously done, with a careful study of these statistics to determine if and how we can improve our results in the future.

The widespread use of neck dissection in otolaryngology is a fairly recent development; therefore, the number of potential "five-year cure" cases is relatively small. This, however, should not deter us from examining our results periodically to gather what information we can from less than five-year cases.

At the Massachusetts Eye and Ear Infirmary, as elsewhere, we have been doing a rapidly increasing number of neck dissections for laryngeal cancer in recent years, and have decided to compile the results to date, both for our own benefit and to contribute to the general fund of knowledge.

This, then, is the purpose of this paper—a statistical evaluation of neck dissections done for laryngeal cancer at the Massachusetts Eye and Ear Infirmary.

The material used in compiling these statistics represents the efforts of many men at the Infirmary, on both the private

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and ward services, and I acknowledge gratefully their permission to use these cases. For the conclusions and opinions drawn from the statistics, however, I shall have to assume the responsibility personally.

This being primarily a statistical paper, I shall avoid any detailed discussion involving the actual technique of neck dissection. This aspect of the problem has been covered extensively and thoroughly in other publications.[1,2] Suffice it to say that it is my feeling and, I believe, the feeling of most men at the Infirmary, that no matter what technique is used, the dissection should be done thoroughly and as extensively as possible. The area covered should be from the trapezius to the midline. All tissue between the platysma muscle and the fascia overlying the prevertebral muscles should be included in the block, with the exception of the carotid artery and the vagus nerve. If necessary, these structures may also be sacrificed unilaterally, realizing the dangers of ligating or removing the common or internal carotid. The inferior portion of the parotid gland below the level of the mandible, the submaxillary gland and the submental region should be included in the resected block. In view of the close proximity of nodes to the XIth nerve, I do not believe it good cancer surgery to spare this nerve; and we have seen no serious disabilities resultant from its removal. In respect to technique, I feel definitely that if a neck dissection is contemplated at the time of laryngectomy, it should be done as a combined *en bloc* procedure, despite the arguments sometimes used that metastasis is an embolic phenomenon and that one can divide intervening lymphatics without significantly decreasing the chances of cure. Although this may be true, why take a chance in this respect when laryngectomy and neck dissection lends itself so nicely to a one-stage procedure?

I shall defer discussion on indications for neck dissection, because the results of our statistics have altered my impressions on the indications for operation and, I hope, will alter yours.

Now, let us get on with the results of the statistics.

These compilations are based on 75 patients having neck dissections from 1948 through mid-1955, accompanying or following treatment of laryngeal cancer. Forty-two patients had been treated by laryngectomy, six by laryngofissure, 25 by combined laryngectomy-neck dissection, and two by X-radiation. There were no operative mortalities.

Chart I shows the age and sex distribution, which is about what would be expected. The predominance in males, of course, represents the predominance of cancer of the larynx in males, a fact which is well recognized. The age distribution approximately matches that of carcinoma of the larynx in general.

AGE

0 - 10
11 - 20
21 - 30
31 - 40 ----- 6
41 - 50 ----- 10
51 - 60 ----- 30
61 - 70 ----- 21
71 - 80 ----- 7
81 - 90
91 -
Unknown ----- 1

TOTAL: 75 Cases

SEX:
Male ---- 72
Female --- 3

Chart I.

Chart II shows the site of the primary lesion and is self-explanatory. Six cases (8 per cent of the total cases) in which the lesion was limited to the true vocal cords had mobility of the cords.

Chart III demonstrates the type of pathology found. It is interesting to note that the pathology, *in the cases in which* this information was available, all showed *squamous* cell carcinoma. The distribution among the *grades of* squamous cell lesions is about what one would expect with the exception of the paucity of lesions in Grade IV; however, when one realizes that grading is very relative and that many of

SITE OF PRIMARY LESION

Epiglottis	
Right	20
Left	17
True Vocal Cord	
(one)	
Right	17
Left	22
Anterior 1/2	35
Posterior 1/2	32
Mobile	7
(both)	
Anterior 1/2	2
Whole	10
False Vocal Cord	
Right	15
Left	19
Both	2
False alone	10
False and True Cords	17
Subglottic Extension	
Right	5
Left	3
Pyriform Sinus	
Right	6
Left	3
Base of Tongue Involved	5
Post-Cricoid Involvement	3

*In 20 cases (27%) primary lesion was limited to true vocal cords only.

Chart II.

TYPES OF PATHOLOGY

Squamous Cell Carcinoma	
In Situ	1
Grade I	11
Grade II	20
Grade III	33
Grade IV	5
Unknown	5

Chart III.

the Grade III lesions would be called Grade IV by many pathologists, this does not assume any real importance.

DIRECTION OF METASTASIS

Primary lesion limited to one side--------56

Metastasis to same side------------47 (84%)
Metastasis to opposite side---------6 (10%)
Metastasis to both sides------------3 (6%)

Chart IV.

Chart IV demonstrates the frequency of crossed metastasis. As can be seen from the chart, 84 per cent of the patients in whom the primary lesion was limited to one side, metastasized to the same side; 10 per cent metastasized to the opposite side, and 6 per cent to both sides. This, again, approximates closely previous thinking regarding the relative rarity of crossed metastasis of laryngeal lesions.

Chart V shows the overall survival rate. It is of interest to note that of those dead of disease, 22 or 65 per cent were dead within one year. The fourth subdivision of Chart V, "those living without evidence of recurrence," represents the cases in which we are most interested. It will be noted that of the total of 75, 47 per cent were living without evidence of recurrence. This figure, however, is one of no real significance since a number of these patients were operated on less than a year prior to the compilation of the statistics; however, when we break down the number living without evidence of recurrence into "potential" cures for the time period stated; that is, take the percentage of those living without evidence of recurrence of the *total* patients operated on over one year or two years, etc., the figures become significant.

From this point I shall take the liberty of dealing in terms of "two-year cures". This is done primarily because the total number of patients does not permit of dealing in five-year cures; but I believe we are statistically justified in dealing with "two-year survival" rates, and that they are a fairly accurate measure of survival for the following reasons. It will be seen in the last part of Chart IV that the percentage of survival in each year grouping from two years on is fairly uniform, and the percentage in each grouping closely approximates the overall average of these percentages; also, our "two-year survival rate" closely approximates five-year survival rates published by others.

SURVIVAL

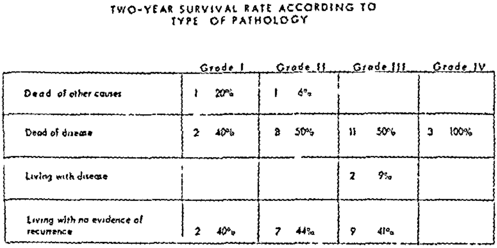
		Per Cent of Total	% of potential "cures" for time period stated	% of "2-year cures
Dead of Other Causes				
Less than 1 year	1	3%		
More than 1 year	0			
More than 2 years	1			
More than 3 years	0			
More than 4 years	0			
More than 5 years	0			
Dead of Disease				
Less than 1 year	22	45%		
More than 1 year	5			
More than 2 years	5			
More than 3 years	2			
More than 4 years	0			
More than 5 years	0			
Living with Recurrence				
Less than 1 year	1	5%		
More than 1 year	2			
More than 2 years	0			
More than 3 years	0			
More than 4 years	0			
More than 5 years	1			
Living without Evidence of Recurrence				
Less than 1 year	7	47%	70%	
More than 1 year	9		50%	
More than 2 years	4		33%	39%
More than 3 years	5		55%	
More than 4 years	3		25%	
More than 5 years	7		44%	
Total Potential "2-year Cures"	4,			

Chart V.

By taking these liberties statistically and speaking in terms of two-year survival rates, we find that of 49 potential two-year cures, 39 per cent were living without recurrence two years or more, or a 39 per cent "survival rate".

From this point on all statistics will be figured on the basis of these 49 cases of potential two-year cures.

Chart VI shows the two-year survival rate according to the type of pathology. By the time we divide these 49 cases into the various categories shown, the numbers become rather small, and are probably not especially significant; however, upon examination of the chart, we see that there is no striking effect on the survival rate by the type of pathology. Thus, if any conclusion can be drawn from this chart, it would be that the grading of the pathological lesion does not materially alter the survival rate.



TWO-YEAR SURVIVAL RATE ACCORDING TO
TYPE OF PATHOLOGY

	Grade I	Grade II	Grade III	Grade IV
Dead of other causes	1 20%	1 6%		
Dead of disease	2 40%	8 50%	11 50%	3 100%
Living with disease			2 9%	
Living with no evidence of recurrence	2 40%	7 44%	9 41%	

Chart VI.

This overall survival rate of 39 per cent compares favorably with previously reported five-year results in neck dissections for cancer of the larynx. Clerf[3] has reported 51 per cent five-year cures; Schall[4] in nine cases between 1931-1948 records 34 per cent five-year cures; O'Keefe[5] reports an overall 42.5 survival rate; and Martin[1] reports 60 per cent five-year survival rate. An average of these percentages yields a 47 per cent five-year survival rate.

I was at first heartened by these statistics, in that our survival rate was within 9 per cent of the average of survival rates of others. It was also heartening to realize that we are curing, by neck dissection, a large number of patients who were previously doomed when metastatic nodes were treated only by radiation; however, in reviewing the records on these patients, I began to notice more and more relationship between the size of the metastatic nodes involved and the survival of the patient. This led me to divide the cases into two groups; one in which, at time of operation, the metastatic node or the largest of the metastatic nodes was 2 cm. or less in size, and a second group in which the nodes were over 2 cm. in size. I then tabulated the two-year survival rate on this basis and was astounded at what I found.

I hope that you, too, will be astounded, because I believe the results are extremely important and demonstrate that we have to change our philosophy, indications for surgery, and management of these patients.

SURVIVAL WITH REFERENCE TO SIZE OF METASTATIC NODES

Living without evidence of recurrence	Metastatic Nodes under 2 cm in size		Metastatic Nodes over 2 cm in size	
More than 1 year	73%		29%	
More than 2 years	75%	Av 87% 2-yr survival	13%	Av 7% 2-yr survival
More than 3 years	83%		0%	
More than 4 years	100%		10%	
More than 5 years	88%		0%	

(% refers to per cent of potential survivors in time period stated)

Chart VII

Chart VII shows these results. You will see that when the node is less than 2 cm. in size, we obtained an 87 per cent two-year survival rate; on the other hand, if the node was over 2 cm. in size, the survival rate was only 7 per cent.

In order to be sure that these statistics were not influenced unduly by those patients who had a combined laryngectomy and neck dissection, I took out the cases which had the combined operation and determined the survival rate on these in the same manner. Chart VIII shows that, when the nodes were under 2 cm. in size, there was an 80 per cent survival rate; when the nodes were over 2 cm., there was a 14 per cent survival rate. Thus, this general ratio of 80 per cent to 10 per cent, depending upon the size of the node, holds true whether the neck dissection is done as a combined procedure or secondarily.

SURVIVAL IN PATIENTS HAVING COMBINED
LARYNGECTOMY-RADICAL NECK DISSECTION

Living without evidence of recurrence 2 years or more "2-year survival	Nodes under 2cm. in size	Nodes over 2cm. in size
	60%	14%

Chart VIII.

I think you will admit that here is a startling difference: 87 per cent survival when the node is small (2 cm. or less) versus 7 per cent when it is larger. These figures cannot be ignored! What does this finding mean in terms of future care of our patients? It means that we cannot be smug about achieving a 39 per cent survival rate when we see that a potential 87 per cent survival rate is possible. It also means that we have been falling down on the job, and it answers my question voiced in the first page of this paper as to whether we are taking full advantage of this cervical node barrier. Most definitely, we are not taking full advantage of it. If we were, our cure rate would be in the neighborhood of 85 per cent instead of 40 per cent. It also means that as doctors we are not detecting and operating upon cervical metastases while they are small enough to have this advantage of an 87 per cent chance of survival. Why is this so? I believe it is due to a combination of factors.

One reason is the tendency to attempt to spare the patient an unnecessary operation. This is, of course, a highly desirable state of mind but not so desirable when it is carried to the point where the patient's chance of cure is cut from 87 per cent to 7 per cent. It has been common practice, when finding a cervical node, to think that the node may be inflammatory rather than metastatic, and to observe the patient for a period of time until one is definitely satisfied that the node is metastatic. This invites disaster, since this period of observation may be the time required for the node to become large enough to fall into the 2 cm.-7 per cent survival class. (We have seen nodes develop and grow to a large size in a period of 2 or 3 weeks, this happening where we are sure that competent observers have seen the patient previously with no palpable nodes.)

Delay has also been due to the reticence on our part to tell the patient to be on the lookout for nodes in his neck. I believe, as a rule, we tend to avoid this for fear of unduly exciting or frightening the patient, or of making him overly conscious of his neck so that he will feel and palpate it excessively.

Another reason we have been falling down on this job is that we, as otolaryngologists, have looked on this procedure of neck dissection as such a formidable procedure and such a big, long operation that we subconsciously attempt to avoid it unless we are absolutely and finally convinced of its necessity.

A further cause of delay frequently overlooked is the difficulty of palpating small nodes in obese patients. Even the most searching and delicate of fingers cannot detect a 1 cm. node under one or two inches of fat.

Now, how are we going to avoid these pitfalls in the future? How should we adjust our thinking to avoid making the same mistakes again? How can we raise our statistics from 40 per cent to the 85 per cent we now know is possible?

First of all, we must be much more meticulous in our frequency of follow-up visits. In the Tumor Clinic of the Massachusetts Eye and Ear Infirmary, the general practice has been that of having the patient return every month for the first post-operative year, every two months the second year, every three to four months the third year, etc., until five years is reached when the checkup is at yearly intervals. It would seem that we should probably tighten up such a schedule a little more and have the patient return every three weeks the first year, and every four to six weeks the second year. At any rate, I think you will all agree that, if we are to detect nodes early, we must see the patients more often. We must also be careful to make our examinations at these visits extremely careful and complete. As pointed out by King,[6] there is a tendency for follow-up visits by grateful cancer patients to become social events. This must be avoided at all costs. We all know how difficult it often is to feel a cervical node when it is less than 2 cm. in size, and these follow-up visits just have to be done as carefully as possible in order to detect nodes when they are small.

We must also start leaning over backwards in the direction of doing neck dissections on suspicious nodes. We must stop waiting too long to determine whether the node is cancerous or inflammatory. Two weeks, at the most, should be the maximum waiting period for determining whether a node is inflammatory or metastatic. Biopsy of the node is one answer to this dilemma; but it is to be condemned, because it is a very good way to seed surrounding tissues with tumor cells, open lymphatics for dissemination of disease, and complicate a later neck dissection by scarring, loss of tissue planes, etc.

Another solution to this problem is the use of frozen-section biopsy, with the patient and the doctor prepared to proceed immediately with neck dissection if the report is positive. This procedure also has its drawbacks in that the cancer bearing region is invaded, and the best opportunity for *en bloc* removal is lost. Thus, except for occasional cases where there is a serious belief that the node is inflammatory, the procedure of choice is to have the courage of one's convictions, decide upon and proceed with definitive neck dissection. Granted that by so doing we will probably do a certain number of unnecessary neck dissections. This operation, however, is not a mutilating procedure; and if we can raise our cure rate from 40 per cent to 87 per cent, then I believe some unnecessary operations are justified. Again, when can we really say that the operation was unnecessary? We may do the procedure and find no positive lymph nodes, but this is no guarantee that the patient was not going to develop metastatic nodes subsequently.

These statistics should also make us less wary of worrying or exciting the patient by asking him to be on the lookout for lymph nodes. We cannot see the patient as often as he sees himself. He should be warned to be watchful for any lumps in his neck and to report immediately any suspicious swelling, no matter how small. Incidentally, this can be done without unduly worrying the patient by telling him that these nodes, or lumps, are probably stitch abscesses or infections and that they should be seen as soon as possible to be effectively treated.

In obese people we have to be extremely careful in neck palpation and should be more inclined towards a neck dissection for the slightest suggestion of an underlying node.

This brings us to the last means of avoiding our previous delays, and that is in the terms of our general outlook on neck dissections. We must stop looking upon the operation as a major procedure to be avoided, if possible. We must develop our skill and ability to perform the operation so that we can and will perform it without undue trepidation. We should have no more compunction about advising this procedure than we would in advising a mastoidectomy or a laryngectomy. We should also develop our technique to the point where we can perform the procedure in a reasonable length of time, so that long operating time will not be a deterrent to advising operation.

I think you will agree that on the basis of the past record, there is much opportunity for advancement and improvement in our management of cervical metastases from cancer of the larynx. This is not surprising, because cancer of the larynx is one type of cancer where, if we do an adequate and thorough job, a very high percentage of cures is possible. Because of its early warning signs, we should be able to detect the primary lesion early; and because of the ready accessibility of the metastatic lymphatic area, we should be able to detect and remove the metastases early. It would seem that it is in this area of detecting and removing metastases early that we have been failing mostly; and it is this area wherein lies our greatest opportunity to increase the overall cure rate for cancer of the larynx.

Fortunately, there is evidence that we are learning from past experience. In listing Dr. Clerf's overall five-year cure rate, I neglected to say that his cases were broken down into three periods. From 1929 to 1940, the cure rate was 14 per cent; from 1940-1944 it was 60 per cent; from 1945-1949 it was 78 per cent. I have no way of being certain, but I believe that this dramatic increase in the cure rate is due to the fact that the operation of neck dissection is being accepted and being undertaken more and more as the years progress, and that it is being done for earlier lesions. Another example of this tendency is in our own institution where, as you can see, between 1948 and the middle of 1955, there were approximately 75 neck dissections performed, whereas I find that in the year 1955 alone, 54 neck dissections were performed.

Because of time limitations, I have purposely avoided the problems of "prophylactic" or "preventive" neck dissection. This is a very provocative question at the present time, but I venture to predict that, in the not too distant future, preventive neck dissection for all extra-cordal cancer of the larynx will be a routine, commonly accepted procedure. After all, we have been discussing the importance of doing neck dissections early, and what better way to do it early than to do it before the metastatic nodes have been developed.

Without doubt, there has been a rapidly increasing interest in neck dissection in our field, and a rapid increase in the awareness of its need. This is represented by the increasing number of articles dealing with the subject in our literature and also in the increasing frequency with which it is being done for primary carcinoma in the field of otolaryngology. I'm afraid, however, that we have been placing more emphasis on how to do the operation rather than upon when to do it.

Surely, we have all known that it is important to do the operation as soon as possible; but here we have, in black and white, figures showing just *how* important early operation is. I hope that these figures will start us all thinking more in terms of when to do a neck dissection rather than how to do it.

SUMMARY.

1. The statistical results of 75 cases of neck dissection done for primary carcinoma of the larynx at the Massachusetts Eye and Ear Infirmary are presented.

2. The overall "two-year survival rate" of potential two-year survivors was 39 per cent.

3. If the metastatic nodes were 2 centimeters or less in size, the two-year survival rate was increased to 87 per cent; whereas, if the metastatic nodes were over 2 centimeters, the two-year survival rate was 7 per cent.

4. These statistics demonstrate the tremendous importance of early detection and removal of cervical metastasis from carcinoma of the larynx.

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DISEASES OF THE SALIVARY GLAND.[*][†]

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INTRODUCTION.

Otolaryngologists often are confronted with the problem o diagnosing and treating an abnormality of the salivary glands These glands are subject to disturbances of development anc function, inflammation and tumor formation, just as are othe structures of the body (see Table I). In the majority of in stances, establishing the diagnosis and instituting the prope treatment are simple matters. Some patients with disease o the salivary glands, however, present problems of diagnosis or management which tax the skill and ingenuity of the examiner.

TABLE I.

CLASSIFICATION OF DISEASES OF THE SALIVARY GLANDS

A Developmental Disturbances (Congenital Anomalies).

B Secretory Disturbances
  1. Sialorrhea· a. Physiologically normal, b. True; c. [illegible]
  2 Xerostomia· a. Idiopathic; b Acquired.

C. Calculus Formation

D. Inflammation· 1. Acute; 2 Chronic

E Tumors: 1. Benign; 2.

F Miscellaneous Condition
  scure etiology; 2 Hyper

with numerous small glands situated throughout the mucous membrane of the mouth which secrete a mucoid material, normally produce between two and three pints of saliva during a 24-hour period. Despite the fact that the parotid gland is the largest of the principal salivary glands, it contributes only 26 per cent of the total salivary secretion. The submaxillary glands, though smaller, produce about two and one-half times, or 69 per cent, as much, while the sublingual glands contribute five per cent.

The secretion of the parotid gland normally is thin and watery while that from the sublingual gland is thick and mucoid. The secretion of the submaxillary gland may be either thin and watery, thick and viscid or a mixture of both, depending upon the nature of the secretory stimulus. The salivary glands are under the dual control of the autonomic nervous system. The parasympathetic control is largely concerned with the secretion of fluid, while the sympathetic division controls the liberation of organic matter from the cells. Experimental evidence suggests that each cell receives fibers from only one of the autonomic divisions, and that the composition of the resulting secretion depends upon the group of cells stimulated and the nature and intensity of the secretory stimulus.

The normal amount of saliva is so constantly present in the mouth under ordinary conditions that we are apt to fail to appreciate its contributions to our comfort and digestion. By means of the act of mastication, saliva performs what may be its most important function: it moistens and converts food taken into the mouth into a plastic mass and envelops it with a lubricant coating so that the food may be easily swallowed. In addition, the sensation of taste is promoted as the saliva dissolves soluble elements of the ingested food. Although food remains in the mouth for only a short time, salivary digestion may account for an appreciable part of the digestion of starch. Ptyalin, which is contained in mixed saliva, is thoroughly mixed with the ingested food during mastication and continues to act upon the starch molecule after the bolus of food reaches the stomach until it is inactivated by the highly acid gastric juices. The constant flow of saliva contributes to oral hygiene by washing away food residues, epithelial cells and foreign material. The removal of such debris by the salivary flow, together with the inherent bacteriostatic action of saliva, tends to inhibit the growth of bacteria in the mouth. In addition, saliva contributes to our comfort by moistening and lubricating the mucous membrane and soft parts of the mouth and lips to keep them pliable so that ingesting food and speaking is made easier.

DEVELOPMENTAL DISTURBANCES.

Congenital abnormalities of the salivary glands are rare. Any conceivable malformation may occur, ranging from complete absence of glands to failure of one or more to develop to full maturity, which could cause fistula formation, obliteration of salivary ducts or formation of cysts. Congenital tumors, such as hemangioma or lymphangioma, may occasionally involve the salivary glands. Aberrant salivary gland tissue is often found in the mouth and lips, and also may be situated in the lacrimal apparatus, eyelids, eyebrows and skin of the face and neck. Like aberrant tissue found in other areas of the body, it may undergo malignant degeneration.

The diagnosis of most of the developmental defects of the salivary glands is often self evident. The symptoms and clinical findings of an hemangioma, a lymphangioma or a fistula of the salivary gland are similar to those of other areas of the body involved by these pathologic states. In other instances, when the diagnosis cannot be established through careful clinical and Roentgen examination, including sialography when indicated, the diagnosis must depend upon exploratory operation.

The treatment of these lesions other than total absence of a gland is best accomplished by an adequate surgical procedure. Radiation therapy, in my experience, has proved ineffective in controlling tumorous congenital lesions, even when the diagnosis has been established by a biopsy. Similarly, sclerosing or electrocoagulation therapy of fistulas has more often than not proved fruitless, and has made subsequent surgical excision more difficult. Excision of every nodule of obscure origin situated beneath the mucous membrane of the lips or mouth is necessary if the diagnosis of aberrant salivary gland tissue is to be made and adequate treatment instituted.

SECRETORY DISTURBANCES.

Sialorrhea.

Excessive salivation, or sialorrhea, may be real or apparent. The latter occurs when an individual does not regularly swallow saliva that is secreted in normal amounts. Most often it is the result of a painful lesion of the mouth or throat, paralysis of the tongue or pharyngeal constrictor muscles and stenosis or obstruction of the esophagus. True sialorrhea is physiologically normal in the infant from about the third month to the end of the first year of life and apparently is related to teething, since it precedes the eruption of the teeth by several weeks and decreases as the teeth appear. Tobacco is used so commonly today that the mild increase in salivary flow initiated by its use must be considered normal.

Pathologic sialorrhea may be the manifestation of many conditions. Diabetes and diseases of the stomach or pancreas are frequently mentioned in the literature as being a cause for sialorrhea. In a rather extensive clinic practice, however, I have yet to encounter sialorrhea, which I could attribute to these conditions. Similarly, in the literature, sialorrhea is commonly attributed to the use of mercury, bismuth, pilocarpine and potassium chlorate, but they are not a common cause of this symptom today, for they are used infrequently in the treatment of disease. Sialorrhea is a prominent symptom of rabies, irritant poisons such as lye or lysol and, on occasion, of acute infectious disease such as smallpox, when it produces ulceration of the buccal mucosa.

Sialorrhea is a symptom most frequently manifest of local disease of the mouth or throat, disease of the central nervous system, emotional stress and nutritional deficiency. Local irritation of the buccal mucosa and tongue from jagged teeth and poorly fitting dental appliances is frequently productive of sialorrhea. This is particularly true of old dentures which have become relatively porous and impregnated with oral secretion and debris. Acute or chronic inflammatory lesions of the mouth such as stomatitis, whether due to Vincent's organisms or not; gingivitis, alveolar abscess and sprue often cause an increase in salivation, which is most apparent during the acute phases of the disease. Sialorrhea is frequently present with tumors of the mouth and pharynx. The increase in salivation under such circumstances is probably both real and apparent as a result of painful swallowing, local irritation and reflex stimulation of the salivary glands. Disease of the central nervous system is occasionally accompanied by troublesome sialorrhea, which probably occurs as a result of reflex activity of the salivary glands or because the patient has difficulty in swallowing. The commonest of these are motion sickness, tic douloureux, myasthenia gravis, paralysis agitans and encephalitis lethargia. Severe emotional stress or strain may be followed by increased salivation. It is also seen in advanced nutritional deficiencies such as scurvy and pellagra, as well as those of lesser severity.

The commonest features seen clinically in patients exhibiting sialorrhea are drooling of saliva from the corners of the mouth and frequent swallowing. When fissuring of the corners of the mouth is accompanied by drooling of saliva and glossitis, a nutritional deficiency should be suspect. Coughing and choking as a result of aspiration of saliva because of inability to swallow normally are prominent symptoms of paralysis of the tongue and pharyngeal constrictor muscles and, in cases of bulbar palsy, tracheotomy may be required to keep the tracheobronchial tree clear of overflow secretions.

A careful history and clinical examination will establish the diagnosis and, thus, the etiology of sialorrhea in the majority of instances. Fluoroscopy of the esophagus may be necessary in the more obscure cases in order to demonstrate whether an esophageal lesion or a disturbance of deglutition exists. This is particularly true when the onset of salivation, coughing and choking has been sudden. Fluoroscopy at such times usually reveals a hold-up and retention of the barium in one or both valleculae and pyriform sinuses, and is indicative of paresis of the hypopharyngeal musculature, which may be a result of a minor vascular accident involving the vagus nerve.

Sialorrhea is treated by management of the underlying disease of which it is a symptom. In addition, astringent mouth washes and the judicious use of small doses of atropine may be helpful in minimizing the annoyance a patient experiences from this condition.

Xerostomia.

Xerostomia, the antithesis of sialorrhea, is the term applied when salivary secretion is absent or markedly diminished in quantity. Idiopathic xerostomia is a rare condition, most commonly seen in elderly women. The onset of this condition is insidious but progressive, and when it reaches the point where little if any salivary secretion is produced, the physician may discover that the lacrimal or nasal glands also produce less secretion. The etiology of idiopathic xerostomia is unknown, but it is my belief that it occurs as a result of some obscure disturbance of the sympathetic nervous system. Once established, idiopathic xerostomia is permanent and irreversible.

Acquired xerostomia is usually temporary. Xerostomia following radiation therapy for malignancy of the nasopharynx, paranasal sinus or the salivary glands, however, may be more or less permanent. Dryness of the mouth is a familiar complaint of public speakers and is purely nervous in origin, since it is present only while an address is delivered. Drugs such as belladonna, chlortrimeton and the opium derivatives, when administered therapeutically for disease, may cause a transitory decrease in the secretion of saliva. The dryness of the mouth, which is a frequent complaint of patients who have severe emotional disturbances, is usually a subjective symptom, but in some instances there may be an actual decrease in salivary secretion. Local inflammation or neoplastic disease of a salivary gland may produce an actual decrease in salivation, but rarely to the point that the patient is aware of it. Xerostomia is frequently associated with severe systemic infections or other diseases which cause either unrecognized or inadequately treated dehydration, such as those of the liver or kidneys, diabetes mellitus, diabetes insipidus, typhoid fever, cholera and systemic viral disease. Such diseases, with the exception of diabetes mellitus, are infrequently encountered in an otolaryngological practice, but should be kept in mind in evaluating any patient who complains of dryness of the mouth.

Patients who are afflicted with idiopathic xerostomia are miserable. The mucous membrane of the mouth and throat is dry and glazed in appearance and the tongue is furrowed, dry and rough when palpated with the finger tip. Clumps of tenacious mucus may be seen on the posterior pharyngeal wall, but no secretion can be expressed from the ducts of the principal salivary glands. Swallowing solid food is all but impossible due to the lack of saliva, and food must be washed down with fluid. Consequently, the diet is usually inadequate with respect to normal nutritional requirements. The appearance of the mouth, tongue and throat in patients with acquired xerostomia is similar to that of idiopathic xerostomia but to a lesser degree. The mucous membrane of the mouth and tongue is moistened to a variable extent by saliva which is sticky in consistency when touched with the finger tip or throat stick. Little secretion can be expressed from the principal salivary ducts and the secretion is more viscid than normal. Some difficulty in mastication and swallowing may be experienced at the beginning of a meal but this decreases, for chewing augments the salivary flow to some degree.

Idiopathic xerostomia, in my experience, fails to respond to any form of treatment intended to increase the secretion of saliva. This is understandable, for sialography demonstrates atrophy of the secretory elements of the principal salivary glands. Consequently, treatment is limited to improvement in the oral hygiene, the use of soothing mouth washes such as those containing glycerin, and supplementary vitamins and feedings to improve the nutritional status. Similar therapy is also beneficial in acquired xerostomia. In addition, augmenting the fluid intake with unsweetened lemonade, chewing gum or sucking a lemon may be beneficial in acquired xerostomia. Nicotinic acid in sufficient dosage to produce mild flushing or a sensation of warmth, and pilocarpine in therapeutic doses to stimulate the salivary glands may increase the salivary flow.

CALCULUS FORMATION.

Salivary calculi are not rare. They occur more frequently in male than in female patients. They are situated most commonly in the submaxillary gland or its duct, probably because the secretion of the submaxillary gland has a greater viscosity and a higher protein content than that of the other salivary glands; the duct is longer, and the body of the gland is situated below the level of the buccal orifice so that the rate of flow of its secretion is slower than that of the other glands.

Calculi are composed primarily of calcium salts and, although their origin is still controversial, investigation suggests that they result from precipitation of calcium salts about a nidus of bacteria or precipitated mucin.

Calculi located in the principal salivary gland ducts produce symptoms characteristic of obstruction of the duct. The onset is most commonly associated with the ingestion of food and is ushered in by sharp pain, followed by progressive enlargement and tenderness of the gland involved. These symptoms usually subside completely following the initial attack. As further attacks are experienced, the pain becomes less intense, the tenderness constant and more pronounced and the swelling of the gland never entirely recedes. Secondary infection in some degree occurs almost invariably with recurrent obstruction, and purulent material can be expressed from the duct. On occasion, a calculus may be extruded spontaneously from a duct. When this occurs, relief is obtained from the acute symptoms of obstruction but may be followed by persistent enlargement of the gland and tenderness when eating, as a result of the development of an inflammatory stricture at the site originally occupied by the calculus.

Calculi within the substance of the gland usually produce similar but less severe symptoms than those of duct calculi. When infection supervenes, these symptoms become intensified. Purulent material may be observed as flakes in clear secretion expressed from the gland or as intermittent clumps of cloudy, thickened secretion alternating with what appears to be normal saliva. When the infectious process involves the parenchyma of the gland, frank pus can be expressed from the duct. If the infectious process is allowed to proceed without interference or progresses in spite of therapy, the development of local abscesses and cellulitis is inevitable.

Calculi located in a duct, particularly when situated near the duct orifice, are easily palpable through the buccal mucosa. When they are situated in the substance of the gland, however, they cannot be palpated unless they are extremely large or are incorporated in the buccal surface of the submaxillary gland. In most instances, the history is characteristic and when coupled with a stone that can be palpated either with the finger tip or a probe in the duct, the diagnosis is certain. When a stone cannot be palpated, Roentgen examination, including an intra-oral occlusal film of the suspected gland and duct, often will demonstrate the calculus. Not all calculi are of sufficient density to be radiopaque, however, and when routine Roentgen examination fails to demonstrate a suspected calculus, sialography will be successful in establishing the presence of a nonopaque calculus or of a stricture.

Calculi in the duct of the submaxillary or parotid gland can usually be removed intra-orally without danger if they are situated completely within the duct. Minute calculi may be expressed from the duct following dilation of the duct with lacrimal dilators. When they are larger and cannot be removed by simple dilatation, a linear incision of the duct, using the largest lacrimal dilator that can be passed as a guide, will permit deliverance of the stone. This latter procedure also serves to rectify partial obstruction of a salivary gland secondary to stricture formation. When larger stones are situated in the duct just after it leaves the gland or are in the substance of the submaxillary gland but are palpable just beneath the mucous membrane of the floor of the mouth, removal through an intra-oral approach is possible but more difficult. When such a situation exists, the calculus must be prevented from being pushed farther into the substance of the gland by passing a traction suture through the mucous membrane and the substance of the gland posterior to the stone. When this has been accomplished, incision of the mucous membrane and gland substance directly over the surface of the calculus will frequently permit its removal with forceps. Only one attempt should be made to remove a stone in this situation because of the possibility of injuring the lingual nerve and spreading infection. If this procedure is unsuccessful, excision of the submaxillary gland and its contained calculus is indicated; furthermore, if multiple calculi are present in the substance of the gland or if calculi reappear in the duct following previous removal, excision of the submaxillary gland is the treatment of choice. Calculi situated in the substance of the parotid gland are usually located near the beginning of Stensen's duct, and can be removed only through an external approach directly over the suspected location of the stone. In every instance in which an acute infectious process is associated with calculus formation, the acute process should be controlled by antibiotic therapy or other measures before surgery is instituted.

INFLAMMATION.

Mumps, or acute epidemic sialadenitis, is the prototype of inflammatory disease of the salivary glands. It is a communicable disease, usually occurring in epidemics, and is characterized by swelling, pain and tenderness of the parotid gland with associated fever. Mumps is caused by a filtrable virus and, although it may affect persons of all ages, it occurs most frequently in patients between the ages of 5 and 15 years. The course of the disease is usually uneventful, but complications may occur. Orchitis rarely develops before the age of puberty, and in adults, during the course of an epidemic, the incidence may be quite high. Meningo-encephalitis may occur and affect patients of all ages. Deafness is occasionally a complication of mumps and is usually irreversible.

During epidemics the diagnosis of mumps is simple. Mumps occasionally involves the submaxillary glands, but when these glands alone are involved it is only during an epidemic that a diagnosis can be established at the time. The blood differential count usually exhibits a lymphocytosis and this, together with complement fixation and skin tests, may be of help in establishing the diagnosis in the absence of an epidemic or in differentiating mumps from an acute sialadenitis of bacterial origin.

Inflammation of the salivary glands of bacterial origin may be acute or chronic. The parotid gland is most commonly involved unless the suppurative process is secondary to a calculus, in which case the submaxillary glands are more frequently implicated because of the predilection of these glands for calculus formation. Acute parotitis, in my experience, has almost invariably been the sequel of dehydration in patients subjected to major abdominal operations or severe debilitating illness in whom a lowered resistance to infection probably has contributed to the establishment of the infection. The route bacteria follow to infect the gland has been considered to be either hematogenous or by direct extension through the duct. It is my opinion that the latter route is the one most often implicated. When a patient is inadequately hydrated it is quite conceivable that the gland, devoid of its protective flow of secretion, may become infected by retrograde extension of mouth flora through the duct, particularly in individuals with poor dental hygiene and septic oral cavities.

Usually only one of the parotid glands is involved by the inflammatory process although, on occasion, both may be affected. The onset is sudden and is usually first noted by the patient as a tender swelling at the angle of the mandible which progressively increases in size and becomes painful. Fever is present and the skin overlying the gland becomes tense, reddened and shiny. Inspection of the mouth reveals the quantity of saliva to be less than normal with the result that the tongue blade used for the examination tends to stick to the mucosa; the papilla of the duct is reddened and pouting. On palpation, the swollen gland is hot, exquisitely painful and, when pressure is exerted from the back of the gland toward its duct, pus is expressed from its orifice.

On occasion, the parotid gland is involved by direct extension of a suppurative process situated in the pharyngomaxillary space. Although the parotid is swollen and painful and pus can be expressed from its duct, these symptoms are less intense than with the previously described inflammatory process. Careful examination of the throat in such cases will reveal edema of the tonsillar area on the side of the involved gland, which is almost pathognomonic of this condition. Treatment is primarily that of pharyngomaxillary space abscess.

The treatment of acute suppuration of the parotid gland which I have found most effective consists of correcting the underlying contributory dehydration and instituting adequate antibiotic therapy. Initially, the antibiotic is given intravenously and thereafter intramuscularly until reports of the culture and sensitivity tests of the purulent secretion expressed from the parotid duct are obtained. If these tests indicate that another antibiotic preparation might prove more effective, it is changed at that time. In addition, hot compresses are applied to the swollen gland and the patient is instructed in "milking" the gland. He is urged to do this several times a day. If the suppurative process appears to respond slowly or inade quately to this therapy, resolution may be hastened by dilating the parotid duct and irrigating the gland with the solution of antibiotic.

Roentgen therapy, in my experience, has been of little if any benefit in treating such cases. On one occasion, the therapy described proved to be ineffective, and surgical intervention was necessary to effect a cure. Although a surgical procedure is rarely necessary, it can be accomplished without danger to the facial nerve and, when indicated, should be instituted without delay. The surface of the parotid gland is easily exposed by reflecting the skin and subcutaneous tissue forward from an incision in the pretragal crease, beginning at the temporomandibular joint and extending inferiorly to terminate 2 or 3 cm. anterior to and below the angle of the mandible. Once the lateral surface of the gland has been exposed, numerous incisions are made in the substance of the gland paralleling the course of the branches of the facial nerve. These incisions in the substance of the gland are then deepened and enlarged with blunt forceps to facilitate drainage. The wound is allowed to remain open and secondary closure effected when the suppurative process has subsided.

In my experience, acute sialadenitis of the submaxillary gland has always been secondary to obstruction of the duct by a calculus. If the stone is accessible and can easily be removed, the suppuration usually subsides rapidly. If phlegmon of the adjacent tissues is associated with the suppurative process of the submaxillary gland, however, medical treatment similar to that described for acute parotitis should be instituted and the more acute phase of the inflammatory process allowed to subside before any attempt is made to remove the stone surgically.

Chronic suppuration of the parotid gland occurs relatively infrequently. When present, however, it proves to be exceedingly troublesome to the patient, and is characterized by acute exacerbations of the suppurative process alternating with periods of relative quiescence. Its etiology is unknown, but failure of the initial acute inflammatory process of the gland to subside completely or the presence of an inflammatory stricture of the parotid duct or its orifice is probably contributory. If the patient is seen during a quiescent stage, the gland may be only slightly enlarged and indurated with purulent secretion mixed with clear saliva expressible from the duct. When the patient is suffering from an acute exacerbation, however, it cannot be differentiated from an original acute parotitis except by the history of similar recurrent attacks.

Sialography always demonstrates some abnormality of the duct or the gland. A stricture of the orifice of the parotid duct is usually discovered only while dilating the duct in preparation for the instillation of lipiodol for sialography. When properly carried out, the sialogram of a patient with chronic parotitis may demonstrate that the duct system of the gland is abnormal in that the principal duct is stenosed or elongated, tortuous and dilated. In others, the parotid duct may appear normal while the smaller ducts are dilated and irregular in outline, with puddling of the lipiodol in the acini.

Treatment of chronic parotitis is difficult and often unsatisfactory. Even when an abnormality of the parotid duct, such as a stricture, can be eliminated, the suppurative process is usually so well established in the gland substance that, although the symptoms are improved, a cure is not obtained. Prolonged antibiotic therapy based upon culture and sensitivity tests frequently causes temporary abatement of the inflammatory process and occasionally results in a cure. Frequent "milking" of the gland and occasional irrigations of the duct to remove accumulated purulent secretion and enhance the salivary flow appear to be helpful in reducing the frequency of acute exacerbations of the infection. When all else fails and the patient demands permanent relief from his symptoms even though mild paresis of the facial nerve may ensue, total parotidectomy will effect a cure.

Chronic suppuration of the submaxillary gland is invariably the result of recurrent calculus formation or of extensive irreversible damage to the gland substance from a previous calculus. The treatment of choice is excision of the submaxillary gland.

TUMORS.

Tumors of the salivary glands are not common, but neither are they rare, since it has been estimated that they comprise one to two per cent of all tumors. Considerable controversy exists among pathologists as to their histologic classification. Although the classification of Foote and Frazell* may not be entirely satisfactory, it is one that is similar to the classification employed by the pathologists at the hospitals in which I operate (see Table II). These neoplasms are predominantly of epithelial origin. Those that are considered to be of connective tissue or other origin occur so infrequently that they will not be considered in this presentation.

TABLE II.
TUMORS OF THE MAJOR SALIVARY GLANDS.*

A. Mixed Tumor: 1. Benign; 2. Malignant.

B. Muco-epidermoid Tumor.

C. Squamous Cell Carcinoma.

D. Adenocarcinoma: 1. Adenoid cystic carcinoma. 2. Miscellaneous forms: a. Trabecular or solid adenocarcinoma; b. Anaplastic adenocarcinoma; c. Mucous cell adenocarcinoma; d. Adenocarcinoma with pseudoadamantine pattern. 3. Acinic cell adenocarcinoma.

E. Papillary Cystadenoma Lymphomatosum.

F. Oxyphil Cell Adenoma.

G. Benign Lympho-epithelial Lesion.

The majority of the neoplasms of the salivary glands are located in the parotid gland and are benign. Clinical differentiation between benign and malignant tumors of the salivary glands is usually impossible preoperatively. Pain occurs much more commonly as a symptom of malignant disease, but cannot be relied upon for differential diagnosis as it may be present, on occasion, when the tumor is benign. Facial paralysis, however, is a positive preoperative indication that the tumor is malignant, for it indicates that neoplastic disease has infiltrated the facial nerve. On one occasion, however, the presence of a facial paralysis was misleading. This patient had a large, rapidly growing tumor which had recurred not long after previous excision, and an associated facial paralysis. The tumor was freely movable, however, which is not usually the case in malignant disease. Careful questioning elicited the facts that the current palsy had begun in a period of 24 hours, and that the patient had experienced a similar attack several years previously from which recovery was incomplete. My preoperative impression that the patient had a recurrent mixed tumor and a Bell's palsy was confirmed at operation.

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* Foote, F. W., Jr., and Frazell, E. L.: Tumors of the Major Salivary Glands. Washington, D. C., Armed Forces Institute of Pathology, 1954. Sec. 4, Fasc. II.

Mixed tumors are the commonest neoplasm affecting the salivary glands, and their adequate surgical management is applicable to all benign tumors of these glands. These tumors are usually round or ovoid and are encapsulated. The capsule is usually thin and delicate, and frequently minute appendages of tumor are attached to it by fibrous strands. Mixed tumors have a marked tendency to recur, and for this reason it is important that every vestige of the tumor be removed at the time of the original operation. The recurrent mixed tumor usually is a poorly defined aggregate of multiple nodules in contrast to the primary tumor, which is almost invariably a solitary nodule. This difference in the gross appearance of recurrent and primary mixed tumors indicates that the former arise from foci of the primary tumor left behind at the time of the original operation. Such foci of recurrence undoubtedly have their origin in the spillage of neoplastic cells when the capsule is inadvertently ruptured, or in the minute excrescences of tumor tissue allowed to remain when a tumor is enucleated. In order to avoid these contributors to recurrent disease, it is necessary that the primary tumor be excised with a wide border of grossly normal salivary gland tissue surrounding it. Every effort should be made to preserve the facial nerve when the lesion is benign. This can be accomplished best at the time of the original operation, for there is greater danger that the nerve will be injured when recurrent tumor masses must be excised.

Malignant tumors of the salivary glands, such as squamous cell carcinoma, do not occur as frequently as benign tumors. They are much more difficult to manage satisfactorily. Biopsy of a salivary gland tumor in order to establish the histologic diagnosis prior to definitive operation is, in general, contraindicated. The first surgical procedure should be instituted with the intention of eradicating the neoplasm. If, at operation, the tumor is thought to be malignant whereas preoperatively it was believed to be benign, biopsy is indicated. If the frozen section unequivocally establishes the neoplasm as malignant, a more radical procedure must be considered. If the accuracy of the frozen section diagnosis is questionable, further definitive surgery should be delayed until control sections have been examined. The extent of a more radical procedure, when indicated, will be determined by the histopathological type of the malignant tumor, since there are wide differences in the clinical behavior of the several types that may be encountered. In general, unless the tumor has spread locally to an obviously inaccessible region or distant metastases are present, the operation should consist of total excision of the salivary gland and a radical neck dissection. When the parotid gland is involved the facial nerve should be sacrificed. There may be instances when all or part of the facial nerve may be preserved, but it must be recognized that such conservation carries a calculated risk. If the facial nerve is sacrificed it may be reconstructed about a week after the original operation, with satisfactory relief from the facial palsy. If there is any doubt that the tumor has been adequately excised, supervoltage radiation therapy is indicated.

MISCELLANEOUS CONDITIONS.

Chronic asymptomatic enlargement of the parotid glands is associated with Mikulicz's disease, Sjögren's syndrome and uveo-parotid fever. The etiology of these conditions is so obscure and they are so rarely encountered in otolaryngological practice that they will not be discussed. Chronic hyperplastic enlargement of one or more of the salivary glands is occasionally encountered, but the cause of the increase in size of the gland is not known. Enlargement of the salivary ducts and glands is seen on occasion in glass blowers and musicians who play wind instruments; such cases are of interest only in that they occur as a result of an occupation. Neurofibromas of the facial nerve, lymphomas and branchial cysts occasionally cause enlargement of a salivary gland. Their differentiation from specific disease of these glands can be established only by surgical intervention.

Lacerations of the salivary glands resulting in fistula formation and facial paralysis deserve mention. When an individual who has suffered a laceration of the face exhibits a facial paralysis or leakage of saliva from the wound, the wound should be explored with the intention of repairing the severed facial nerve and the salivary fistula. This is best accomplished at the time the laceration is originally sutured. Salivary fistulas which are seen after the laceration has healed are best eradicated by excision of the fistulous tract. The resultant defect in the salivary gland is ablated by suturing the gland together, without drainage. In my hands, this has proved a more satisfactory form of treatment than radiation therapy for the management of salivary fistulas. Delayed repair of the severed facial nerve is always tedious and often difficult, but the result that can be obtained more than justifies the labor involved.

SUMMARY.

The major salivary glands not uncommonly exhibit disease. In the majority of instances this is due either to a disturbance in its development or function, or to involvement by calculus formation, inflammation or neoplastic process. Occasionally it may be the result of tumors arising from the nerves, blood vessels or lymphatics contained in the gland, or in rare instances it is a manifestation of an obscure condition of unknown etiology such as Mikulicz's disease, Sjögren's syndrome or uveo-parotid fever. In addition, a salivary fistula and paralysis of the face may be the residual of a facial laceration which involved a salivary gland.

Congenital anomalies are rare and are best treated by surgical intervention when necessary. Significant secretory disturbances of the glands occur infrequently, but on occasion sialorrhea may be a prominent symptom of local disease of the mouth or paralysis of the vagus nerve. Xerostomia, whether idiopathic or acquired, is a troublesome condition, and its response to treatment frequently is unsatisfactory. Calculi occur most commonly in the submaxillary gland or its duct. They should be removed surgically through an intra-oral or external approach, depending upon the location of the stone and gland involved, but when recurrent or multiple calculi affect the submaxillary gland excision of the gland is the treatment of choice. Acute inflammations occur most frequently in the parotid gland and usually respond satisfactorily to adequate hydration and antibiotic therapy. Chronic suppurative disease, on the other hand, often responds indifferently to medical measures. Surgical intervention may be required to remove an obstruction to the flow of saliva or to remove the entire gland in order to effect a cure.

Tumors of salivary gland origin are either benign or malignant, and their histopathologic classification is somewhat controversial among pathologists. The treatment of these tumors is adequate surgical excision. The parotid gland is most frequently involved by neoplastic disease and, in the majority of instances, the tumor is benign. Benign lesions, such as mixed tumors, should be removed with a wide margin of grossly normal tissue surrounding the tumor in order to minimize recurrence. Enucleation of such tumors is to be condemned, and is contrary to the tenets of adequate tumor surgery. Malignant tumors require radical excision. As a rule, malignant disease of the salivary glands necessitates complete removal of the involved gland, immediately adjacent tissues insofar as possible, and a radical neck dissection. No attempt should be made to preserve the facial nerve except in rare instances, as it can subsequently be reconstructed to avoid a permanent facial paralysis.

PRESENT STATUS OF THE OPERATION FOR MOBILIZATION OF STAPES.*†

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Considerable interest has developed in the surgery of the stapes and oval window since Rosen[1] described the operation for the mobilization of the stapes in 1953. Direct manipulation of the stapes to relieve ankylosis of the footplate was attempted by several investigators at the end of the last century and, although there were reports of some good results, the operation was abandoned for some undisclosed reason. Considerable skepticism was aroused when surgery of the stapes was reintroduced in a modified form.

Until recently few otologists would grant that the stapes could be mobilized when fixed by otosclerosis; others believed that once mobilized the stapes would quickly become fixed again. Some otologists suggested that middle ear adhesions and perforations of the ear drum would be frequently encountered and would interfere with hearing. It was also suggested that following the operations for the mobilization of the stapes the patient would be an unsuitable candidate for other rehabilitative procedures.

Answers to many questions in regard to this surgery can be found in this study of 100 cases observed at the Manhattan Eye, Ear and Throat Hospital for a period of almost two years. Although we have found sufficient grounds to provide answers to some of these anticipated questions many new and still unsolved problems have developed. In attempting to determine the present status of this surgery we can say that a significant contribution has been made in redirecting the interest of modern otologists toward the restoration of hear-

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ing by surgical procedures on the stapes; however, our studies indicate that there are many uncontrollable factors in the present technique and further investigation may bring forth a new and more reliable method of dealing with the stapes and oval window.

By attempting to discuss the commonly asked questions and express our opinions concerning the problems we have encountered, we believe we can accurately evaluate this surgery as it stands today and substantiate our views concerning the status of the operation for the mobilization of the stapes.

How often can hearing be improved by manipulation of the stapes?

Using the technique and instruments described by Rosen, the mobilization of the stapes operation was performed on 100 patients. In these cases the canal was incised, and the tympanic membrane was elevated out of the annulus and reflected anteriorly. The mobilization procedure was performed by applying pressure on the anterior surface of the neck of the stapes in line with the stapedius muscle tendon. In this series of cases (see Fig. 1) it was possible apparently to mobilize the stapes in 63 of the 100 patients. Thirty-five were definitely fractured, and in two cases the stapes was found to be mobile.

Fig. 1

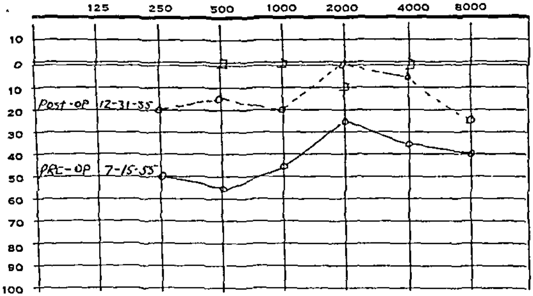
MOBILIZATION OF THE STAPES
100 CASES

Stapes Mobilized	63
Stapes Fractured	35
Stapes Found Mobile	2

Can hearing be improved to the serviceable level by successfully mobilizing the stapes?

Although mobilization of the stapes was considered successful in 63 of 100 cases, hearing was restored to a serviceable level in only 35 patients. All cases were considered to have good preoperative cochlea reserve and fulfilled the criteria used in accepting patients for the fenetration operation. In some patients the postoperative improvement in hearing was quite remarkable and approached the normal. This improvement was noticed immediately by the patient at the time of surgery. In all cases which had a considerable improvement in hearing, the stapes was always found solidly fixed in the oval window and yielded suddenly when sufficient pressure was applied to the neck of the stapes. The stapes obtained a degree of motion which approached the normal. The Rinné test was found to change from negative to positive in the operated ear as soon as the stapes was released from the restraining lesion. Examples of what can be achieved under the most favorable conditions are the case histories of the following patients (see Figs. 2 A and B).

Fig. 2 (Case A).

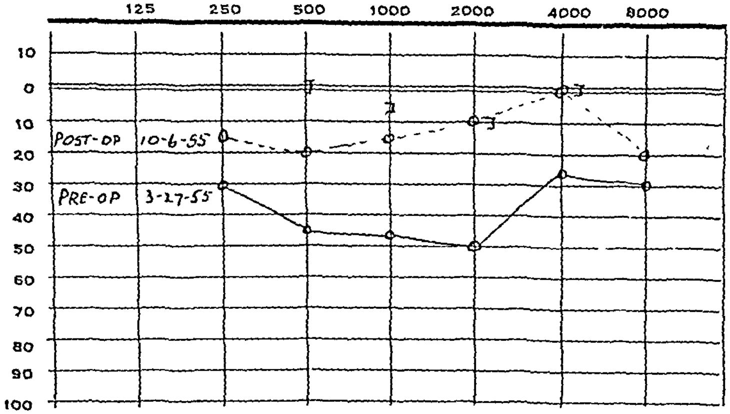


S. R. T.	Preop.	Postop.
Right	45 db	15 db

Right Mobilization, September 26, 1955.

Case A (W. I. L.): A thirty-three-year-old telephone operator who had impaired hearing for six years decided in favor of the mobilization operation which was performed on Sept. 26, 1955. The stapes was found fixed and upon manipulation became suddenly mobile. The Rinné test changed from negative to positive, and the patient experienced an immediate improvement in hearing to the 15 db level for speech.

Fig. 2 (Case B).



S. R. T. 45 db Postop.
Right Preop. 15 db
Right Mobilization, March 29, 1955.

Case B (NAC): The preoperative and postoperative audiograms of this thirty-six-year-old female who had been hard of hearing for eight years show the degree of hearing improvement possible. The stapes was found solidly fixed at the operation performed on March 29, 1955. Upon mobilization the patient experienced an immediate improvement in hearing to this high level of 15 db for speech. The hearing improvement has been maintained to date.

A satisfactory initial improvement in hearing of approximately this degree was obtained in 35 of the 63 patients successfully mobilized (see Fig. 3). Cases which followed this course of behavior were the most satisfactory as the hearing improvement was usually maintained in the postoperative period. Only six patients in this group showed any signs of regression to the preoperative level. This change was apparent in five patients within two months and after five months in the remaining case. Twenty-eight of the 63 cases,

Fig. 3.
MOBILIZATION OF THE STAPES
100 CASES

Satisfactory Initial Improvement	35
Improvement Lost Within Two Months	5
Improvement Lost Within Five Months	1
Hearing Maintained Over Six Months	29
Unsatisfactory Initial Improvement	28
Stapes Mobilized	63

however, apparently mobilized, did not obtain the expected improvement in hearing.

Having apparently mobilized the stapes successfully, why should there be no improvement in hearing in 28 of the 63 cases?

At the time of surgery the conclusion that the stapes was mobilized successfully was based on three unreliable signs of stapedial footplate liberation. *1.* Before manipulation, except in two cases, the stapes was found fixed when palpated. After a successful mobilization procedure the stapes became free, and movement could be seen in the stapedius muscle tendon. *2.* The subjective improvement in hearing which follows immediately after mobilization of the stapes is a definite sign of restoration of stapedial function. There is no question when improvement in hearing is substantial as this change is unmistakable to the patient and the surgeon. When improvement is not considerable this sign of stapedial footplate liberation was found to be less reliable. In one case the stapes was definitely fractured and the fractured crura were removed. While in the operating room this patient maintained that her hearing was definitely improved Subsequently she found no change in hearing, and audiometric examination showed that hearing in the operated ear was at the preoperative level. This demonstrates the unreliability of the immediate subjective response as a sign of a successful mobilization. *3.* It was, therefore, necessary to employ some objective hearing test in the operating room to assist the surgeon in determining whether the stapes was mobilized. A sterile 512 tuning fork has been used successfully to determine changes in stapedial fixation. Before manipulation the Rinné was always negative, being heard better by bone conduction than air conduction. When the stapes was successfully mobilized the Rinné test became positive as it was heard better by air conduction than by bone conduction. When the stapes was fractured the Rinné test remained negative. Many patients could not decide at the time of the surgery whether improvement in hearing had occurred. The decision to continue further manipulation was an important one for the surgeon. The tuning fork was found to be a valuable aid at this point in the operation. At times the Rinné would not become definitely positive but could be heard equally well by air conduction as by bone conduction. This indicated to the surgeon that a change had occurred in stapedial fixation and no further manipulation was necessary; however, all of these are secondary and unreliable signs of what has occurred at the footplate of the stapes or in the crura.

The technique followed in this study is in most cases a blind procedure in which the surgeon has no opportunity to modify his technique according to the pathology encountered or to evaluate the results of the manipulation. It has been possible to visualize part of the footplate of the stapes in only 10 per cent of the cases when the incus and crura of the stapes remains undisturbed. In most cases a small part of the posterior crus was seen and only rarely could the surgeon visualize any part of the more significant anterior crus. As great variation occurred in the position of the middle ear structure, it was found difficult at times to determine the exact angle assumed by the crura. Careful study of these poorly visualized structures, however, affords the surgeon some help in determining in which direction to apply the mobilizing force.

Many cases assumed to be mobilized successfully may have developed green-stick fractures of the crura or a variety of similar injuries. By palpating the neck of the stapes these injuries could be misinterpreted as a successful mobilization of the footplate. A rather large group of patients, 28 of 63, considered successfully mobilized at the time of surgery did not obtain the expected improvement in hearing. The poor results obtained in this group must go unexplained as we have no knowledge of the pathology encountered or what changes occurred as a result of manipulation.

Poor visualization of the region of the oval window prevents exact evaluation of another clinical observation which may be important. By palpation it was possible to detect three different types of stapedial fixation. *1.* The stapes was solidly fixed without any resiliency and defied considerable pressure. It was assumed that this type of fixation was caused by a massive otosclerotic extension which involved the footplate and anterior crus of the stapes. Sufficient visualization was possible in one case where the lower half of the anterior crus and anterior portion of the footplate were seen to be involved in a large otosclerotic mass. Fracture of the crura occurred in almost all cases of solid fixation when mobilization was attempted according to the method used in this study.

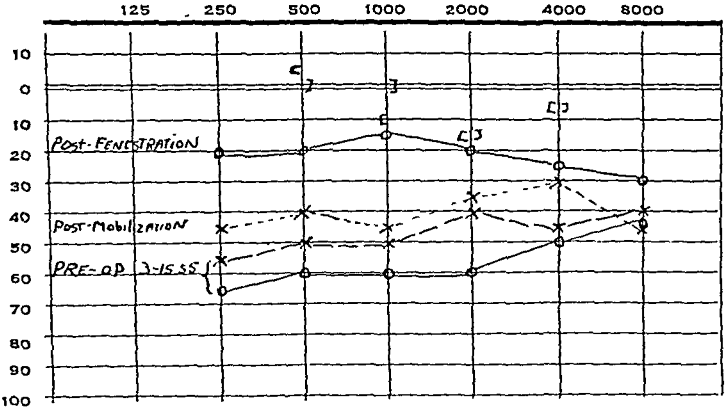
2. In the second type of fixation the stapes was found to be solidly fixed but yielded to moderate pressure on the neck. There was complete and sudden mobilization which left the stapes liberated, and movement of the head was possible by very slight pressure of a probe. In all cases of this type there was definite subjective improvement in hearing. The Rinné test immediately became positive. It is reasonable to assume that the otosclerotic lesion in this type of fixation was a limited and narrow filament of bone binding the footplate to the oval window. The stapes could be freed under these circumstances as the restraining lesion could be overcome with less force than was necessary to fracture the crura.

3. In other cases the stapes was often found to be firmly but not solidly fixed. An impression was obtained that fixation was not caused by a solid lesion. Attempts at mobilization were productive as the stapes was mobilized, but it did not become completely and freely movable as previously described. There was a residual "set in rubber" feeling when the stapes was palpated. At the time of surgery the patient experienced a hearing improvement, and the Rinné test may have become equal or may have remained negative. The improvement in hearing was maintained usually for several hours but disappeared within a day or two. Again we have no means available to examine the region of the footplate and crura in order to determine the basic pathology present at the oval window which could be responsible for this type of behavior.

An instrument resembling a nasopharyngoscope is now being developed which may be of assistance in visualizing the pathology around the oval window. With it we may be able to understand the factors responsible for the poor results following an apparently successful mobilization. Our experience, therefore, with the mobilization procedure has revealed that the liberation of the fixed stapedial footplate follows three patterns of behavior. We plan to study in more detail the responsible factors involved in each pattern.

Examples of cases apparently mobilized successfully but without the expected improvement in hearing are as follows (see Figs. 4 A and B):

Fig. 4 (Case A).



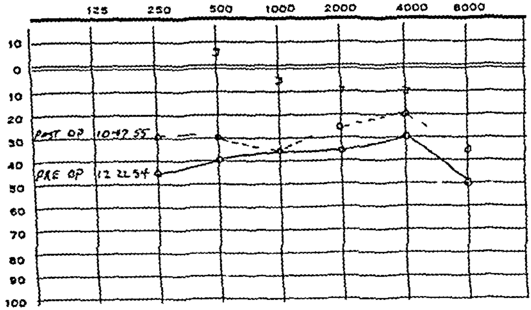
S. R. T.	Preop.	Postop.
Right	60 db	20 db
Left	55 db	50 db

Right Fenestration, April 21, 1955.

Left Mobilization, September 28, 1955.

Case A (HAL): This 32-year-old female presented a history of impaired hearing of ten years' duration. She wore a hearing aid constantly until she had a fenestration operation on April 21, 1955 when her hearing improved to the 20 db level for speech. A mobilization was performed on the left ear on Sept. 28, 1955. At the time of surgery the patient was not sure that an improvement of hearing had occurred, and the Rinné test remained negative. The stapes was found fixed and was considered to be mobilized successfully; however, the ossicle did not obtain the degree of freedom characteristic of the better result. On returning to her room the patient stated that she noticed a moderate improvement in hearing, but this lasted only several hours. Subsequent audiometric tests reveal that a slight improvement in hearing had occurred; however, the Rinné test had always remained negative. This slight improvement in hearing was considered to be far less than was expected, and this patient was listed with the unsuccessful results. The basic pathology and underlying stapedial behavior demonstrated by this case is not entirely clear.

Fig 4 (Case B)



S R T	Preop	Postop
Right	45 db	40 db

Right Mobilization January 28 1955

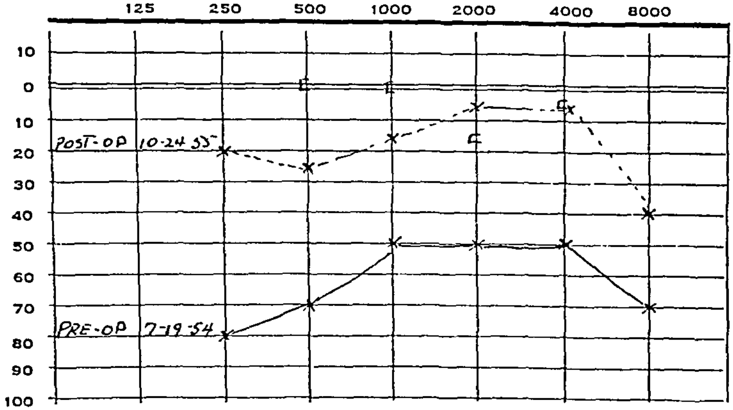
Case B (DeA) A 34 year old male with a history of impaired hearing for five years A sufficient cochlea reserve was found, and a mobilization operation was performed on the right ear The stapes was found fixed and mobilized without difficulty It however, did not obtain sufficient mobility, and at the time of surgery it was questionable whether any improvement in hearing had occurred The Rinné test became equal as the patient heard equally well by air conduction as by bone conduction Upon arriving at his room he was aware of improved hearing for several hours This improvement was lost by the next day and did not return

Is there any correlation between the audiometric findings and the ability to mobilize the stapes?

In this study there was no consistent preoperative observation which could indicate the possibility of success or failure of the mobilization procedure. Patients with severe conductive hearing loss were sometimes mobilized as easily as those with a moderate loss (see Figs 5 A, B and C)

In Case A, therefore, we can assume that the otosclerotic lesion was a limited one, although it was solid and offering complete fixation The crura in this case were strong and able to withstand the pressure required to free the stapes from the restraining otosclerotic lesion In Case C the otosclerotic lesion was assumed to be broad and the crura of the stapes weak The preoperative audiogram, therefore, has not been of as-

Fig. 5 (Case A).

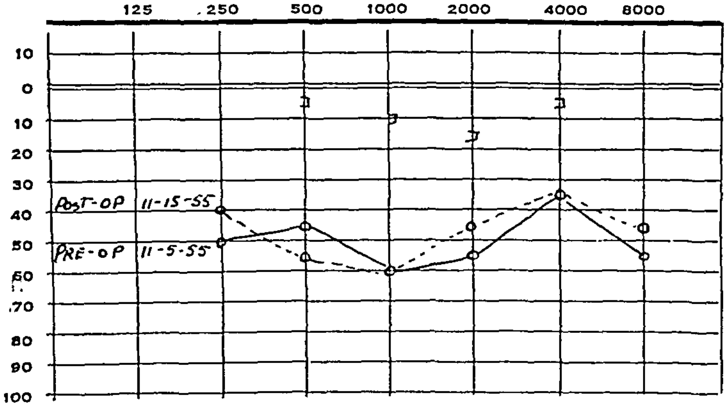


S. R. T. Preop. Postop.
Left 65 db 20 db

Left Mobilization, August 11, 1954.

Case A (MAR): This patient presented a severe preoperative conductive hearing loss. The stapes was found solidly fixed and yielded to moderate pressure. It suddenly became free, and the patient experienced a remarkable improvement in hearing which has not changed for over one year.

Fig. 5 (Case B).

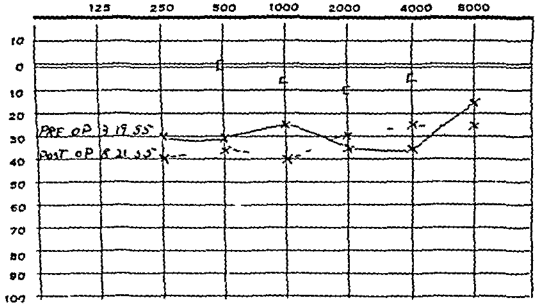


S. R. T. Preop. Postop.
Right 55 db 55 db.

Right Mobilization, November 8, 1955.

Case B (ZAN): In this patient a lesser conductive loss was recorded than that demonstrated in Case A. At the time of surgery it was possible to visualize the anterior crus and footplate of the stapes. These structures were fixed in a large otosclerotic mass. The crura of the stapes were large and strong; but in applying pressure to the neck of the stapes the crura were fractured, and no improvement in hearing resulted.

Fig 5 (Case C)



S R T	Preop	Postop
Left	40 db	45 db

Left Mobilization April 2 1955

Case C (McD) This patient presented a still less conductive loss than the previously cited cases and *was not successfully mobilized as the crura of the stapes was found to be thin and unable to stand sufficient* pressure to liberate the footplate from the restraining lesion Although it was not possible to visualize the footplate and anterior crus in this case it can be assumed that a broad otosclerotic mass prevented successful mobilization

sistance in determining which cases could be successfully mobilized and which would result in failure

How can the conductive hearing loss be explained in the two cases found to have mobile stapes?

It is indeed impressive to find that preoperative criteria for conductive hearing loss, in specific ossicular malfunction, is so often reliable In this series of 100 cases the stapes were found fixed in the oval window in all but two cases In one case, although all the preoperative hearing tests were indicative of a conductive hearing loss, no improvement in hearing followed the surgery The stapes was found very mobile, no adhesions in the middle ear were present, and no pathology was visible in the region of the round window

The stapes was found mobile in one other case There was, however, considerable amount of fibrous adhesions in the region of the round window. When this was removed a flat promontory was found, and no niche was present at the round window. This area was replaced by dense fibrous tissue. No improvement in hearing followed the attempts to remove these adhesions and to manipulate the stapes.

Is there any significant relationship between the duration of deafness and age of the patient with the ability to mobilize the stapes?

It has been found possible to mobilize the stapes of older patients who have had hearing impairment for many years, and the crura have been fractured in relatively young patients with hearing impairment of short duration (see Fig. 6). The upper and lower limits of age and duration of hearing loss of patients who have had improvement of hearing following the mobilization operation were found to be not statistically different from those cases in which the stapes was fractured. This investigation, however, reveals that those patients who have had a satisfactory improvement in hearing following the mobilization procedure were as a rule younger and had loss of hearing of a shorter duration than cases in which the crura of the stapes were fractured. We can conclude that the possibility of successfully mobilizing the stapes exists regardless of age and duration of deafness; however, there is a statistical advantage for patients who are younger with a relatively short history of impaired hearing.

Fig. 6.
MOBILIZATION OF THE STAPES
100 CASES.

		Stapes Mobilized	Stapes Fractured
Age of Patient	Upper Limit	57 yrs.	66 yrs.
	Average	30.2 yrs.	38.0 yrs.
	Lower Limit	19 yrs.	21 yrs.
Duration of Deafness	Upper Limit	14 yrs.	21 yrs.
	Average	5.6 yrs.	11.1 yrs.
	Lower Limit	1 yr.	1 yr.

Is tinnitus influenced by a successful mobilization of the stapes?

Contrary to the general impression, preoperative tinnitus was experienced by only 48 per cent of the patients studied (see Fig. 7). A successful mobilization does influence the tinnitus as 50 per cent of the successfully mobilized patients who had experienced improvement in hearing either lost tinnitus or it was considerably diminished. One patient stated that roaring tinnitus disappeared after a successful mobilization of the stapes; however, approximately three months later the tinnitus returned suddenly; from that time she noticed the improvement began to diminish, and the audiogram gradually approached the preoperative level. When the stapes was not mobilized there was no change noted in the character and intensity of the tinnitus.

Fig 7

TINNITUS

Incidence of Tinnitus	Per Cent
Present	48%
Absent	52%
Successfully Mobilized Stapes Tinnitus Relieved	50%
Unsuccessfully Mobilized Stapes Tinnitus Unchanged	100%

Have any complications occurred following the mobilization of the stapes operation?

In this series of 100 operations the drum was injured in 14. A small perforation somewhere in the posterior half of the ear drum was the usual type of injury encountered. These injuries were found to heal quickly and completely. To date there are no perforations which remain unhealed.

Twelve cases have had postoperative infection following mobilization of the stapes. This has not been a problem to control as the infections responded readily to antibiotics. There is no case in this series which developed a chronic otorrhea, and none have required prolonged postoperative care of any kind.

Vertigo was experienced for a few minutes by several patients during the mobilization procedure. This generally occurred when the stapes suddenly became free from its fixed position One patient complained of vertigo and vomiting after leaving the operating room [illegible] hour. There have been no labyrinthine disturbances of long duration.

Temporary facial paralysis was encountered in one patient when too much anesthetic solution was injected into the external auditory canal. The solution entered the region of the parotid gland and anesthetized the facial nerve. This paralysis lasted three hours. Following this technique no surgical injury to the facial nerve can occur; however, this surgery deals with the area of the facial nerve, and cases are on record where permanent facial paralysis have resulted from the mobilization procedure. The surgeon is impressed by the wide variation in the position of the middle ear structures. These variations are of continuing interest for the experienced otologist, but also provide hazards to the inexperienced surgeon.

Having obtained a satisfactory improvement in hearing following a mobilization of the stapes operation, is the improvement maintained?

Satisfactory initial improvement in hearing occurred in 35 of the 100 patients studied. In these cases the stapes was found to be fixed, and manipulation restored movement to a degree which compared favorably with the normal. In the majority of cases this improvement in hearing has been maintained; however, our investigations have not extended over a sufficient length of time to provide accurate statistics regarding stability of these cases.

Fig. 8.
MOBILIZATION OF THE STAPES 100 CASES

Maintenance of Hearing Improvement

Length of Observation	Number of Cases
Over 18 Months	4
Over 12 Months	9
Over 8 Months	13
Over 6 Months	3
Total Cases over 6 Months	29

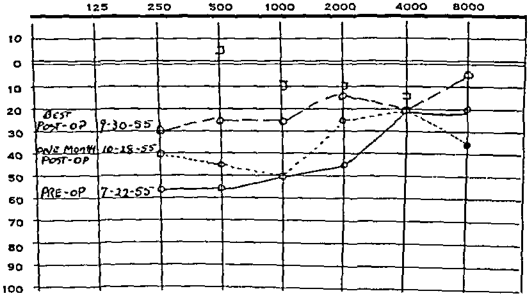
Our observations to date are summarized as follows: As the first operation at the Manhattan Eye, Ear and Throat Hospital was performed on April 14, 1954, no case in this series has been observed for a period longer than 19 months.

Cases which had a satisfactory improvement in hearing are grouped (see Fig. 8) with respect to the number of months the hearing improvement has been maintained.

Four cases have maintained hearing improvement at the original level for over 18 months; nine over 12 months; 13 cases over eight months, and 3 over six months. Of the 35 patients who obtained an initial improvement in hearing to a satisfactory level, six showed signs of regression and returned to the preoperative level of hearing. Five cases lost the initial improvement within two months and only one patient, having survived this two-month period, lost the improvement in hearing five months postoperatively. It can be assumed, therefore, that the improvement in hearing following the mobilization of the stapes is usually maintained if the initial improvement was not lost within a period of two months. Having survived the two-month period, only one patient returned to the preoperative level.

Typical of the cases which were successfully mobilized with a satisfactory initial improvement in hearing and subsequently lost this improvement within two months, is the following case history (see Fig. 9 A). The only case which maintained

Fig. 9 (Case A).

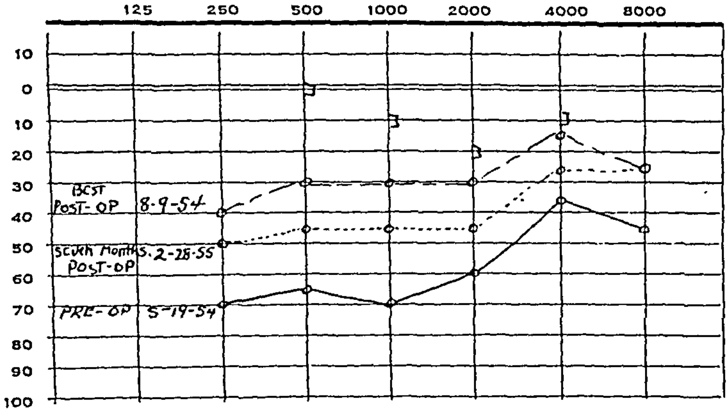


S. R. T.	Preop.	Best Postop.	Last Postop.
Right	50 db	25 db	40 db

Right Mobilization, September 22, 1955.

Case A (ZAD): A 35-year-old female presented a history of impaired hearing of four years' duration. A right mobilization operation was performed on Sept. 22, 1955. At the time of surgery the stapes obtained adequate mobility, and the Rinné test changed from negative to positive. The patient experienced a satisfactory subjective improvement in hearing, and the speech reception threshold was recorded at 25 db level. An audiogram performed on Oct. 28, one month postoperatively, when the patient noticed a drop in hearing acuity, revealed that regression had occurred, and the speech reception threshold was found to be 40 db.

Fig. 9 (Case B).



S. R. T.	Preop.	Best Postop.	Last Postop.
Right	55 db	30 db	45 db

Right Mobilization, July 21, 1954.

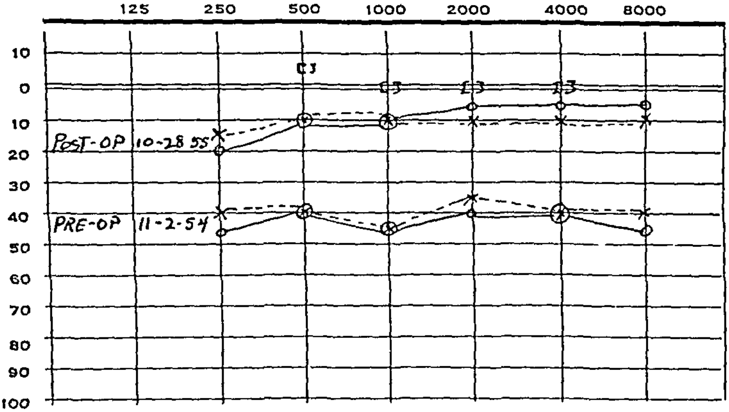
Case B (ARP): This patient presented a history of unilateral deafness of two years' duration. A mobilization operation was performed on the right ear on July 21, 1954. The stapes was found fixed and was successfully mobilized. The Rinné changed from negative to positive, and the patient experienced a satisfactory improvement in hearing. Postoperatively she was able to locate the source of sound without difficulty. Five months after the surgery the hearing in the operated ear became less acute and at present is almost at the preoperative level.

good recovery for 5 months and then lost the obtained improvement is illustrated in Fig. 9 B.

When the stapes does not obtain sufficient mobility at the operation, as was found in 28 of 63 cases, the slight improvement in hearing experienced in the operating room or during the first few days after the operation was not maintained, and the hearing quickly returned to the preoperative level.

Fig. 10 (B).

Case GRA.



S. R. T.	Preop.	Postop.
Right	50 db	15 db
Left	50 db	15 db

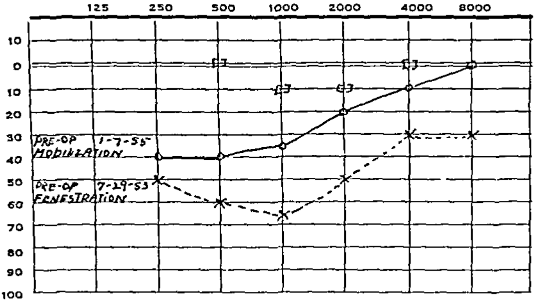
Right Mobilization, June 24, 1955.

Left Mobilization, November 5, 1954.

stapes operation. This is the record of a 29-year-old female who had been hard of hearing for fourteen years and wore a hearing aid continuously. A mobilization was performed on the left ear, Nov. 5, 1954, and the same procedure was repeated on the right ear, June 24, 1955. Hearing has been improved in-both ears to the 15 db level for speech.

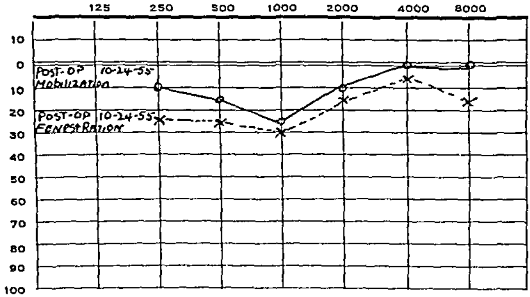
Fig. 11 shows the audiogram of a 25-year-old female who had the fenestration operation performed on the left ear on Oct. 21, 1953. The mobilization procedure was performed on her right ear on Jan. 10, 1955. Although the preoperative conductive hearing loss was found to be much greater in the left ear, the side on which the fenestration operation was done, the bone conduction was considered to be equal in both ears. Both operations yielded satisfactory improvement in hearing, and it is possible to compare the degree of hearing acuity obtainable from each procedure under similar conditions. Although the postoperative improvement by audiometry appears to be approximately equal in both ears the patient states that the right ear, having had the mobilization procedure, is the better at the present time.

Fig. 11.
Case BON.



S. R. T.	Preop.
Right	30 db
Left	50 db

Right Mobilization, January 10, 1955.
Left Fenestration, October 21, 1953.



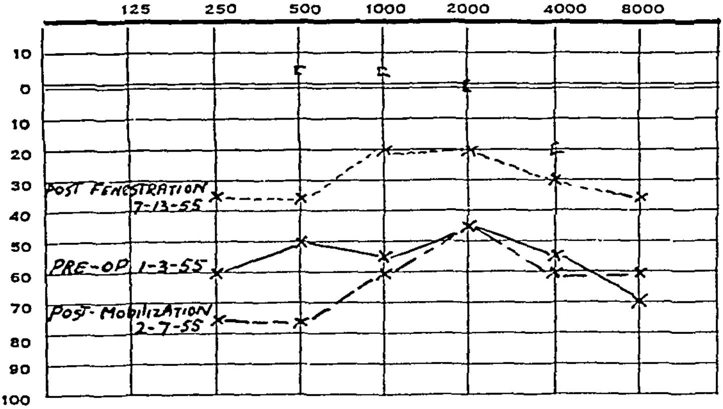
S. R. T.	Postop.
Right	20 db
Left	30 db

It can be concluded that improvement in hearing to a very high level is more often obtained with the mobilization of the stapes operation when individual cases are considered. In this study, however, these good results were obtained in 29 per cent of the cases attempted. With the fenestration operation, hearing is usually improved to the 20 to 25 db level, but in suitable cases hearing has been restored to this level in 70 per cent of our cases accepted for surgery.

Is it possible to perform a successful fenestration operation following an unsuccessful mobilization procedure?

The fenestration was performed on two patients following an unsuccessful attempt to mobilize the stapes. In both cases the stapes was found firmly fixed, and manipulation caused fractures of the crura with no improvement in hearing. The initial incision for the mobilization has been made routinely more superficial than orginally described. Anticipating that

Fig. 12 (Case A).



S. R. T.	Preop.	Post Mob.	Post Fen.
Left	50 db	60 db	25 db

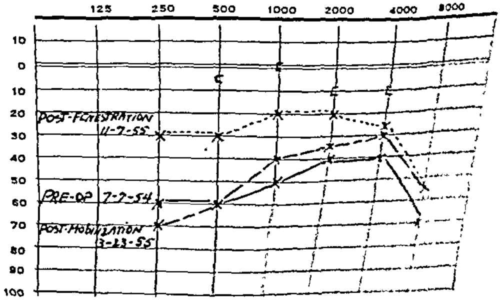
Left Mobilization, January 26, 1955.
Left Fenestration, April 28, 1955.

Case A (NOL): A 23-year-old male who presented a history of hearing impairment of two years' duration which was becoming progressively worse. He was considered an excellent candidate for rehabilitative surgery, and a mobilization of the stapes operation was attempted on Jan. 26, 1955. The stapes was found solidly fixed, and the crura were fractured. On April 28, 1955, the fenestration operation was performed with a satisfactory improvement in hearing.

the fenestration operation would be necessary as a secondary procedure in some cases, the incision in the skin of the external auditory canal was made approximately 8 mm. from the drum. This would provide a sufficiently large scar-free tympanomeatal flap to cover the fenestra. In both cases sufficient time elapsed after the mobilization operation for complete healing of the drum and skin of the external auditory canal. Three months elapsed in one case and eight months in the other.

Both cases were found to be no more difficult technically than a primary fenestration operation. The only signs in the middle ear of the previous surgery were a few thin fibrous strands in the region of the long process of the incus and an irregularity in the annular bone which had been previously removed for better visualization of the stapedial muscle tendon. Both patients had a normal postoperative course and a satisfactory improvement in hearing (see Figs. 12 *A* and *B*).

Fig. 12 (Case B).



S. R. T. Preop. Post Mob. Post Fen
Left 59 db 54 db 25 db
Left Mobilization February 9, 1955.
Left Fenestration October 24, 1955.

Case B (KWI): A 28-year-old female with a history of hea
ment of four years' duration. She was considered a good c
rehabilitative procedures, and on Feb 3, 1955, a mobilization
operation was attempted. The stapes was found solidly fixe
were fractured. On Oct. 24, 1955, the fenestration op..
formed on the same ear. A satisfactory improvement in
and no unusual technical difficulties were encountered . .

Should the mobilization operation be routinely attempted before fenestration surgery?

Although the fenestration operation was found to improve hearing to the serviceable level in 70 per cent of our cases attempted, the advisability of performing the mobilization procedure as a primary operation has become a serious consideration. It has been sufficiently proved that the mobilization procedure has few complications and is able to improve hearing to a high level. The ear drum and canal require no lengthy postoperative care; however, with the technique commonly in use today satisfactory improvement in hearing has been possible in only 29 per cent of the cases we attempted. Very often, therefore, the patient and surgeon must be prepared to face disappointment.

Recently I have had the opportunity to discuss the choice of surgery with one patient who had been hard of hearing for many years. She relied heavily on her hearing aid and had become discouraged when she was told that nothing more could be done for her hearing. Not entirely satisfied, she decided to have another hearing examination. She was found to be an excellent candidate for rehabilitative surgery, and a fenestration operation was performed. She obtained satisfactory improvement in hearing and discontinued the use of the hearing aid. Several months later a mobilization operation was attempted on the other ear. The stapes was found to be solidly fixed, and crura were fractured; no improvement in hearing resulted. It was at this point that I questioned the patient concerning both types of surgery. She admitted that she would have been reluctant to accept the possibility of failure at the time of her first operation; however, knowing well what was involved in both surgical procedures and having spoken to several patients who had obtained excellent results following the operation for the mobilization of the stapes, she voluntarily agreed that for the discomfort involved and the result possible, she would accept the mobilization procedure as the primary step in the rehabilitation program. This discussion crystalizes the position which I believe is most acceptable to the patient and surgeon. Understanding the degree of rehabilitation possible and the relatively small degree of economic and physical handicap imposed by the mobilization of the stapes operation, I do not hesitate to recommend this surgery as a primary procedure; however, I believe all facts concerning both operations should be presented to the patient clearly and frankly. At the present time, the mobilization operation is not a substitute for the fenestration surgery, and I believe that it should not be presented as the only means of rehabilitation available to the patient. Both operations can be presented to the patient as part of a plan of rehabilitation leading ultimately to improved hearing.

SUMMARY.

1. In this study, the mobilization of the stapes operation was found to improve hearing in 29 per cent of the cases attempted.

2. Hearing was improved to a very high level by the mobilization of the stapes procedure as the drum and ossicles are allowed to remain in their physiological relationship.

3. In 28 per cent of the cases the stapes, although apparently mobilized, did not obtain satisfactory mobility; consequently the patient did not obtain a satisfactory improvement in hearing. The basic pathology and physiology responsible for these results needs further investigation.

4. No significant correlation could be established between audiometric findings and the ability to mobilize the stapes.

5. Pathology at the round window can account for conductive hearing loss which can resemble ankylosis of the footplate of the stapes. Two cases were encountered in this series, in which the stapes was not found to be ankylosed.

6. Although there appeared to be no definite influence of age and duration of deafness in the ability to mobilize the stapes, satisfactory hearing improvement has been obtained more often in younger patients with a shorter history of impaired hearing.

7. Only 48 per cent of patients in this series had preoperative tinnitus. Successful mobilization eliminated the tinnitus in 50 per cent of the patients successfully mobilized.

8. No serious complications have been encountered in this series of cases.

9. In this study, postoperative hearing improvement has been maintained for as long as 18 months. Five cases lost the initial improvement within two months and one after five months.

10. The fenestration and mobilization operations can restore hearing to approximately the same level when best cases of each are compared. Excellent results have been obtained more frequently with the mobilization procedure. More consistent good results, however, are obtained with the fenestration operation.

11. Following the mobilization of the stapes operation the fenestration operation was found not to be more difficult.

12. Due to the high percentage of failures following the mobilization of the stapes operation, careful consideration of the patient is necessary before this surgery is recommended.

13. In its present stage of development, the mobilization of the stapes operation should be done as the primary procedure in most cases; however, both operations should be presented to the patient as part of a plan of rehabilitation leading ultimately to improved hearing.

CONCLUSION.

Although excellent individual results are possible when using the present technique for the mobilization of the stapes, the large percentage of failures is principally due to poor visualization of the stapes and oval window. A better understanding of the pathological process involving these structures could be the basis for the development of better instrumentation and technique which seem necessary to perform this surgery with a higher degree of success.

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SEROUS OTITIS MEDIA.*†

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The term *serous otitis media* is used to call attention to the presence of a sterile fluid in the middle ear cavity, the tympanic end of the Eustachian tube and, not infrequently, in some or all of the mastoid air cells. The condition may be acute, subacute, or chronic. In consistency the fluid in the tympanum may be thin and serous, mucus and stringy, or it may be present in the form of a thick, tenacious, gelatine-like mass.

The presence of such fluid in the middle ear cavity and its extensions often presents difficult and perplexing problems in the management of these cases. Chronicity magnifies the problems. I am sure we all have seen a considerable number of patients with fluid in the ear—chronic cases, who had sought help early in the course of their disease. Their ears had been examined; tuning fork and audiometric tests had been performed; the Eustachian tube had been inflated, often with marked but temporary improvement of their symptoms (an occurrence which strongly suggests the presence of fluid). A diagnosis of a blocked Eustachian tube had been made; they were advised that treatment consisting of tubal inflations was necessary. This procedure alone was then carried out over a varying period of time until the patients finally became discouraged and stopped treatment, or sought help elsewhere.

Others may have consulted an otolaryngologist only to be told that they had a nerve deafness for which nothing could be done, and were advised to get a hearing aid. In some cases the patients may have had repeated experiences similar to the above before an accurate diagnosis was finally made. Hoople[14,15,16] and Rawlins[25] have called attention to the fre-

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quency with which the diagnosis in these cases is missed, often by well trained otolaryngologists.

The primary purpose of this discussion is to stress the diagnosis and treatment of serous otitis media and to present the thoughts and procedures of various authors in the management of these cases.

Anatomy: At this time I shall briefly review the anatomy and histology of the parts involved in this disease. The Eustachian tube, the tympanic cavity, the mastoid antrum, and the mastoid air cells are joined to form a continuous group of air-containing channels and spaces. In the adult the Eustachian tube is approximately one and one-half inches long; it is composed of an infero-medial membrano-cartilaginous portion roughly one inch in length and a supero-lateral portion about one-half inch long. It constricts at the junction of the two parts to form the isthmus which, according to Eggston and Wolff,[9] varies greatly in size in different individuals. They also state that the tube is relatively higher, shorter, smaller, and more distensible at the isthmus in the infant. Under normal conditions it is opened regularly and periodically with each act of swallowing or yawning, by the action of the tensor veli palatini muscle. The mucous membrane lining the tube is covered with ciliated columnar epithelium; it contains many seromucinous glands of mixed type near its pharyngeal end: there are no glands in the bony portion of the tube. According to Robison,[26,27] lateral pharyngeal lymph nodes are in close relationship with its cartilaginous portion.

The membranes lining the middle ear and mastoid air cells are very delicate, and in the adult there is relatively little submucosal tissue. In the middle ear the epithelium is of the simple cuboidal type, while in the mastoid it is of the simple squamous type. The nerves, blood vessels and lymph channels traverse the submucosa; the vessel walls are extremely delicate. There are no glands in the submucosa of the middle ear or mastoid; normally not even goblet cells are present in the mucosa, but according to Eggston and Wolff they may appear in the presence of infection. When one considers the histologic and anatomic structure of the Eustachian tube, tympanum, and mastoid, it is clearly apparent that, in the presence of a

not be overstressed. Many of the cases that have gone undiagnosed would not have been missed if the otolaryngologist had taken only a few minutes to listen to the patient's complaints and had taken a history of the case. The onset of symptoms is always sudden.[32] One day the patient hears; the next day he does not. One or both ears may be involved. Almost invariably the patient complains of impaired hearing and a stopped-up ear. Autophony is a common symptom with the patient describing his voice as sounding abnormally loud, echoing into the affected ear, or sounding as though he were talking in a well or in a barrel; he may hear or feel the fluid move in the ear on changing the position of his head; he may complain of his ear or head feeling full, heavy, blocked, or stopped-up on the affected side, or he may comment on the unnatural sound produced by combing or brushing the hair, or by gently manipulating the skin about the auricle on the affected side. If the ear is only partially filled with fluid. he may state that changes in the position of the head, tilting it forward and to the side, result in marked improvement of the hearing and relief from the discomfort, only to have the symptoms recur when the head is in the upright position (according to Eagle[8] these symptoms are pathognomonic of fluid in the middle ear). Often on arising in the morning, the patient may be almost free of symptoms, but the deafness and discomfort soon return as the fluid reaccumulates in the ear. Tinnitus and vertigo are inconstant symptoms.[11,12]

Young children seldom complain of fluid in the ear; a few may complain of head noises. Inattention due to impaired hearing and irritability are common symptoms in children.

Diagnosis: The presence of fluid in the ear should always be suspected from the history. Once the condition is suspected, a careful examination of the ears should definitely establish the diagnosis.

On inspection the drum membrane is never normal in appearance: it is usually retracted; rarely it is bulging; frequently it is scarred, thickened, and opaque; occasionally it may be red and inflamed following an acute otitis media and the administration of antibiotics. A chalky white malleus is frequently seen in long standing cases.[2,5,7,22,35] Rarely does one see a partially filled tympanum with a fluid line, a meniscus or air bubbles; much more frequently the ear is completely filled and, if the drum membrane is fairly normal and translucent, it may have a faint yellow appearance due to the amber-colored fluid. In many cases with a full tympanum the drum membrane will present a dull silver-gray or blue-gray color with a glistening appearance. Rarely one will see a membrane that presents a dark slate-blue color;[30,32] this appearance indicates involvement of long standing. Davison[5] and Hotchkiss[17] have noted greatly decreased motility of the drum membrane in the presence of fluid when using a pneumatic otoscope. As an aid in diagnosis, gentle inflation through a catheter and auscultation is advised by many.[6,22,14,17,24,32]

Hearing tests may prove helpful but, on the other hand, they may be misleading. Invariably, there is some hearing impairment. The hearing loss may be mild or severe; if the disease is not superimposed upon a pre-existing nerve deafness, the hearing loss will be of the conductive type. The Weber test will lateralize to the affected ear; one must not be led astray when hearing tests reveal a nerve type deafness.

If the history of the case, the appearance of the drum membrane, auscultation, and the hearing tests suggest the presence of fluid in the middle ear. but a definite diagnosis still cannot be made, a diagnostic puncture is mandatory.[5,15,22,29,32] This can be accomplished without anesthesia and with little or no pain; even children will often submit to repeated punctures. The ears should first be inflated, preferably by the Politzer method. I prefer to have the patients lying down; in this position they are less likely to move the head and cause pain. The opening should be made through the light reflex at right angles to the radiating fibers;[33] in this position the opening is more likely to remain patent; if fluid is present the opening should be extended and inflation or mild suction may be necessary reveal the presence of fluid.

Prognosis: On the whole. the prognosis is good
short duration. Ears containing a small amount
spond better to treatment than do those filled with
containing a mucus type fluid are usually more
treatment than those containing serous fluid. The

poor in cases where the hearing is improved little or none after evacuation of the fluid. Persistent tinnitus after the removal of fluid warrants an unfavorable prognosis.[24] Regardless of the duration of the condition, the prognosis should not be too gloomy, providing the patient experiences a marked improvement in hearing and relief from all other annoying symptoms following the evacuation of the fluid from the tympanum. It is wise to explain to all of these patients clearly and definitely at the time of the diagnosis that the condition may be difficult to manage, and that several treatments may be required to effect a cure.

Treatment: In the treatment of serous otitis media the objective is to rid the middle ear and its extensions of fluid, prevent its recurrence, and restore and preserve the hearing. Maximum success in attaining this objective can be expected only when one carefully considers and corrects, if possible, the underlying etiologic factor or factors; in rare cases this is an insurmountable problem. Time does not permit further discussion of this important phase of management in these cases.

Local treatment of the condition should be adapted to the individual case. In many cases of short duration where a fluid line is present and the fluid is thin, as shown by rapid changes in position when the head is tilted forward and backward, inflation alone may suffice.[28] Inflation should be repeated several times at each visit. I prefer the Politzer method. The procedure should be done with the head tilted so that the affected ear is up. Inflation should be repeated at daily intervals. If the condition does not subside within a few days a paracentesis is indicated. If the fluid has been present for a week or longer, if it is thick and mucoid, or if it fills the tympanum, an immediate paracentesis is indicated.

There seems to be considerable reluctance on the part of many otolaryngologists to open an ear as a therapeutic measure for this condition. Politzer,[21] in commenting on the procedure, stated that he "considered the operation, after an abundant experience, to be one of the most practically important and successful therapeutic expedients in diseases of the ear."

Suehs states that the operation is contra-indicated in children in the presence of an allergy or hypertrophied and diseased adenoids until the offending etiologic factor has been brought under control or removed. Inflation should precede the procedure; the initial opening should be made through the light reflex and, if possible, it should be extended downward and backward. To evacuate the fluid completely several inflations by the Politzer or Valsalva methods should be carried out at intervals of a few minutes, with the head tilted to the affected side. Before leaving the office, the patient should be instructed to inflate the ear several times daily by the Valsalva method if this can be accomplished; the head should be tilted so that the affected ear is downward. Rawlins and Perlman[23] have developed techniques that enable patients to practice self-Politzerization. Self-inflation is a most useful procedure and should be much more extensively used; it delays closure of the opening, facilitates drainage, relieves congestion in the tympanic mucosa, maintains aeration, and hastens recovery.

A tympanum filled with a thick, gelatine-like mass may present a most difficult problem. In order to evacuate such a mass, one must make a long incision through the drum membrane and apply strong spot suction, using a suction tip with a large lumen. If this fails, several inflations, preferably by the Politzer method followed by spot suction, may succeed. It is sometimes helpful to make a counter opening high in the drum membrane.[23] In such cases, Rawlins, after making a free incision in the drum membrane, uses mass suction with simultaneous inflation. Evacuation of such a gelatine-like mass in children may require a general anesthetic.

Frequently, following a free incision of the drum membrane and thorough evacuation of the fluid from the tympanum, the patient goes on to complete recovery; however, continued inflations are indicated at stated intervals until one is convinced that an early recurrence is not likely. If the opening closes and fluid recurs, repeated myringotomies, usually at intervals of three to five days, are indicated.

Now comes the question: What is one to do with those patients who fail to respond favorably to the usual indicated treatment? Jones,[10] in a few cases, has found an autogenous vaccine made from cultures from the nasopharynx helpful. Budetti and Compere[2] have used ACTH with good results in a few cases that presented clinical symptoms of vasomotor rhinitis. Robison, Theobald,[32] and Armstrong[1] advise making a semipermanent opening in the drum membrane and describe their techniques. Armstrong inserts a piece of small plastic tubing 1.5 mm. in diameter into a myringotomy opening and leaves it in place from one to five weeks; he reports satisfactory results with no complications in a small series of cases. Hotchkiss advises making a small 2 mm. permanent opening in the tympanic membrane in selected cases; he uses a modified corneal punch for creating the opening.

In cases where symptoms persist because of intramembranous exudate in the region of the umbo, Wible[34] reports good results following a free incision through the affected part. Eagle reports good results when use of the Eustachian catheter for therapeutic inflations is discontinued. Deep X-ray or radium treatment of the nasopharynx in the presence of diseased lymphoid tissue in or about the Eustachian tube is recommended by some.[26,27]

As a last resort, numerous authors advise a simple mastoidectomy in selected cases.[5,7,13,26] Jervy,[18] Lawson,[21] Cody,[4] and Suehs discuss the indications for mastoid surgery in these cases.

Comments and Summary: Serous otitis media is a common disease that presents many perplexing problems; diagnosis of the condition is frequently missed. Politzer, as early as 1867, recognized and ably described the condition as a definite disease entity. The anatomic and histologic structures of the middle ear and its adnexa render them susceptible to changes that result in blocking of the tube, creation of a vacuum, and transudation of fluid into the tympanum and its extensions.

Acute and chronic infections involving the tissues of the upper respiratory tract are the most common etiologic factors in the production of the disease. In chronic recurrent cases, allergy is the most common underlying cause.

The diagnosis should be suspected from the history. Careful examinations of the ears, including a diagnostic puncture, should confirm the diagnosis. If the history, symptoms, and the usual examinations do not enable one to make a definite diagnosis, a diagnostic puncture becomes mandatory.

The objective in the treatment of serous otitis media is to rid the middle ear and its adnexa of fluid, prevent its recurrence, and restore and preserve the hearing. Attainment of this objective requires careful consideration and correction, if possible, of the underlying cause or causes. Local treatment of the condition should be adapted to the individual case. To effect a cure, one must evacuate the fluid from the middle ear and restore the normal function of the tube. Satisfactory evacuation of the fluid in most cases is best accomplished by myringotomy followed by inflation. When inflation can be accomplished by Politzerization, this is the method of choice. In rare cases a simple mastoidectomy may be indicated; even this procedure is not always successful in effecting a cure.

CONCLUSIONS.

1. Serous otitis media is a common disease which, if permitted to become chronic, may produce irreversible changes with permanent and disabling deafness.

2. Diagnosis of the condition is frequently missed, even by well-trained otolaryngologists. The history should lead one to suspect the diagnosis. Careful ear examinations, including a diagnostic puncture, should confirm the diagnosis.

3. Maximum success in the management of this condition can be expected only when one carefully considers and corrects, if possible, the underlying etiologic factor or factors combined with adequate, well-chosen treatment of the local condition.

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AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Home Study Courses

The 1956-1957 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, which are offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Detailed information and application forms can be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minnesota. Registrations should be completed before August 15.

PROCEDURE FOR HIGH DORSAL RHINOPLASTY.*

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Philadelphia, Pa.

Jacques Joseph,[1] of Berlin, is often referred to as the father of modern plastic surgery. It was he who originated the method whereby the shape of the nose is altered by making all incisions inside the nose, resulting in no outside scarring. He displayed his inventive genius by devising a complete set of instruments in order to carry out this new approach. The Joseph technique is the accepted method for the correction of nasal deformities, and while minor improvements and individual changes have been recorded, the basic principles of this technique remain unchanged.

Doctors Safian,[2] Brown and McDowell[3] have called attention to the high dorsal nose; otherwise the literature records scant information regarding this nasal deformity.

It is a well-known fact that the angle of the nose on profile, ranges from 25 to 35 degrees. A sharp increase in the angle is productive of a high dorsal nose. This is not to be confused with a large hump nose, where removal of the hump and reducing the nose proportionately in all its dimensions will afford a nice result.

The high dorsal type of nose in the main may or may not have a hump, is large in all dimensions, and the angle is acute, 45° *or more*, which is the dominant factor in the surgical approach.

In the high dorsal type of nose, the entire profile line must be lowered (see Fig. 1) in order to attain the desired esthetic effect to harmonize with the rest of the facial features and stature of the individual (see Fig. 2). As a rule high dorsal noses are long and must be shortened (see Fig. 3). Here again, the shortening must not be severe, since an over-corrected naso-labial angle is not only unnatural but also

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* From St. Joseph's Hospital, Philadelphia, Pa.

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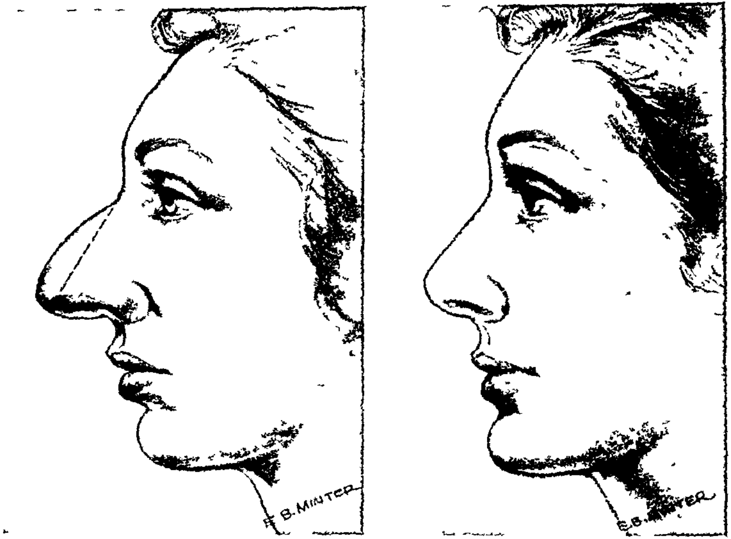


Figs 1 and 2 Sketch showing the before and after correction of the high dorsal type of nose If the high dorsal technique is properly accomplished the results produce a major change Oddly enough most of these noses are not the property of tall individuals so that the same basic principles must be kept in mind—i e —remodeling the nose to fit the *facial features and stature of the individual* conspicuous, and carries the stamp of a nose that has bee corrected. When this excessive tilt is observed on a hig dorsal nose that has not been properly reduced in size, t nose presents a "bird-beak" appearance (see Fig. 4). Th trading of one deformity for another, prompted the writ of this article.

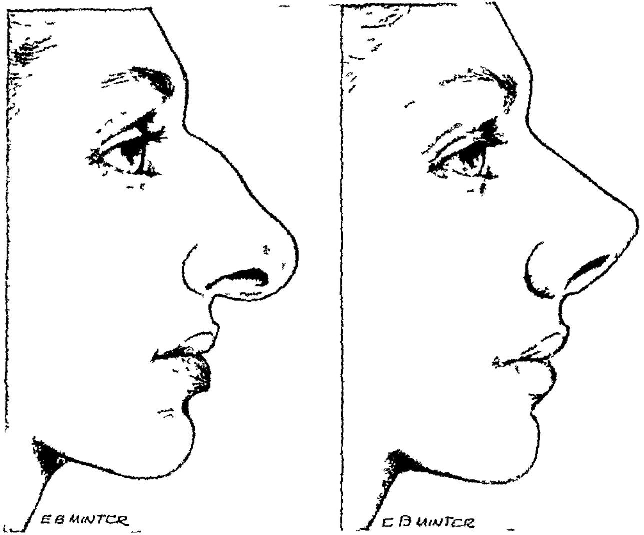
The profilometer first described by Joseph, and late many others, serves no useful purpose. Even those advocate its use rarely if ever employ this measuring d On occasion, having observed demonstrations of the ometer, it appeared quite clear that if the dorsum of th were reduced to the indicated measurements, a re nose would eventuate.

Making sketches to give the patient a mental p what to expect is risky, to say the least. Sketches life size; therefore, one must operate to a scale using a slide rule or some other mathematics. One excellent sketch artist, but is he so exacting in the room where anything may occur and does? Too m are involved concerning the end-result of any wherein regeneration involves the various type No one can boast of 100% perfect results. An extensive experience in this field will attest t so a good sketch artist will sooner or later wish employed this method.

An ordinary wooden tongue blade placed a of the nose (with a little pressure) will giv fairly accurate picture of how the dorsal li in the particular individual. Even then, amount is removed at the time of operati same result will follow—a retruded nose. I has not been sufficiently reduced, the ras the desired effect. This is a simple proce of better results.

Infiltration anesthesia is the accepted procaine with 1-50,000 epinephrine hyd

a full five minutes after th rewards not only

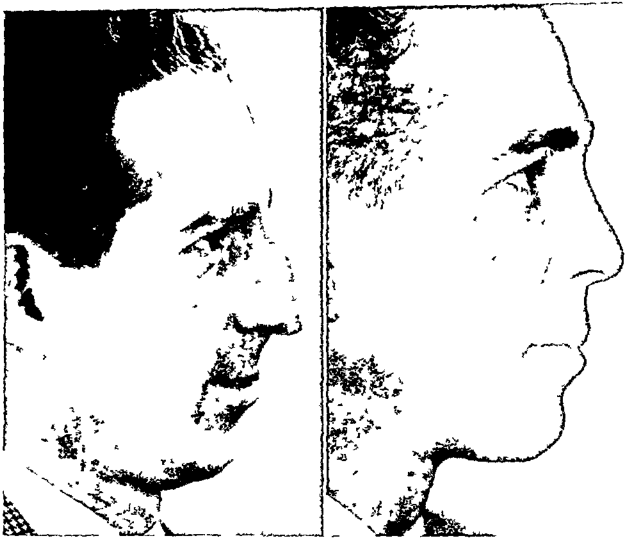


Figs 3 and 4 Medical artists composite drawing of high dorsal type noses improperly corrected It is quite apparent that the dorsal line has only been slightly reduced while the shortening of the nose is overcorrected—presenting a bird beak appearance the patient; bleeding is reduced to a minimum, and there is practically no pain.

Shortening the nose and narrowing the bridge is accomplished in the usual manner so well-known to all who perform these corrections. In reference to the latter procedure, we have utilized the numerous methods hitherto described. Our preference is the orginal Joseph method of using the right angle saws for narrowing the nasal bridge. While the chisel method is quicker, with less effort on the part of the surgeon, the sawing will fracture in a straight line, a method not so accurate when the chisel is used.

Of course every step in a rhinoplasty is important and requires strict attention to details; but reconstruction of the nasal tip demands the most stringent attention. Here again, numerous instruments have been devised, so that an exact amount of alar cartilage is removed from each side. Experience teaches that rarely are the alar cartilages of the same size or shape, even though they may appear so from the exterior; therefore, the removal of the same amount of alar cartilage on each side will not produce the desired effect. The removal or resection of a specific amount of the alar cartilage on either side according to the needs, will in the final analysis produce nares of more equal size and shape; moreover, in a great many instances "splitting" the alar cartilages at the dome is all that is necessary, and in some cases the alar cartilages require no alteration whatsoever. Any over-correction about the nasal tip will produce a pinched appearance. This, with a high tilted nose is certainly not natural. though frequently seen.

Many types of dressing have been employed after the operation has been completed. Metal splints, dental compounds, a combination of both, various dressings and strappings, all have their advocates. Our preference has been to employ the Joseph nasal brace after a small gauze dressing is tailored to fit the nose. The thumb screw can be adjusted so that the brace fits snugly and again adjusted when the patient is seen later in the evening. Should there be considerable swelling, or the brace become loose, it can be adjusted to the required needs with the thumb screw. This we believe



Figs 5A-B Nasal fracture superimposed on a profile elevation The entire profile line was lowered and followed with a *complete* correction *If too much* or too little were removed the desired esthetic effect would not have been obtained A *straight profile line* is the request of most patients especially when they have large noses regardless of the type The *naso-labial* correction increases the length of the upper lip and restores the almost hidden columella to be superior to any other method, since immobilization can be specifically controlled. The brace is worn for two days in the average case—dressings are changed daily. A drop of sealskin* placed on the leather portion of the brace will secure its position. If this is not available, transparent nail polish will answer the same purpose, as suggested by Dr. Edward T. Litt, of Miami Beach, Fla.

We have never found any indications for a turbinectomy. If the mucous membrane about this structure is passively engorged, it can be shrunk to normalcy by numerous methods. If the turbinate still produces obstruction, outward fracture will produce the desired effect; furthermore, a turbinectomy is apt to result in a dry nose.

Before leaving the operating room the patients are instructed not to move the upper lip and if talking is necessary, to limit the conversation to a few words in the manner of a ventriloquist. In addition thereto, if they must sneeze they are instructed to force the sneeze through the mouth in a coughing manner. If the above instructions are adhered to, post-operative bleeding is reduced to a minimum and regeneration is hastened.

These post-operative instructions to the patient are exceedingly important, and a careful search of the literature does not reveal their having been recorded before.

During the period of regeneration, in primary rhinoplasties, a certain small percentage of cases will heal with an excess of fibrous and granulation tissue. This resultant distortion is most commonly observed about the nasal tip and is naturally disturbing to the patient and the surgeon.

It was reasoned that if Cornbleet[4] was able to produce gross shrinkage of keloids to three-fourths of their orginal size we might employ the same agent, Hyaluronidase,†[5] as a preventive measure in the production of excess fibrous and granulation tissue.

The mixture we now employ is 75 units of the Wydase added to 18 cc. of 1 per cent novocaine with adrenalin 1-50,000.

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* Sealskin—Liquid plastic adhesive. Clay-Adams Co., New York.

† Hyaluronidase (Wydase) was furnished through the courtesy of Wyeth, Inc., Philadelphia, Pa.

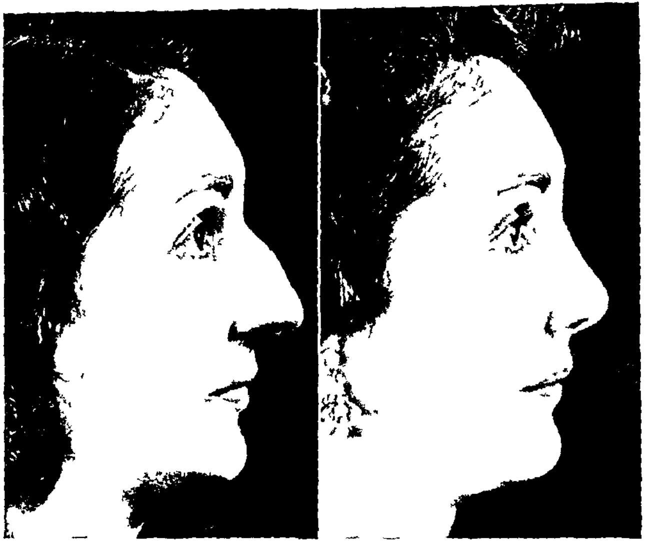

Fig 6A-B Typical high dorsal nose *Removal of only the hump would have resulted in a large nose therefore the* entire profile line must be lowered Narrowing the bridge slight shortening and tip correction are carried out to complete the rhinoplasty

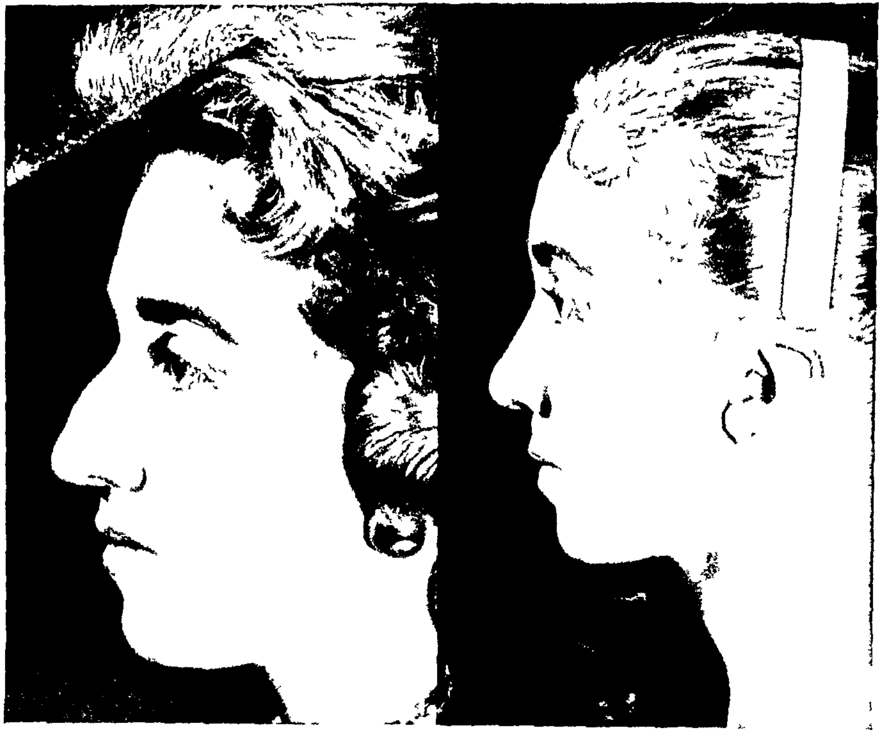

Fig. 7A-B On this congenital high dorsal nose the routine procedure for correction was followed The dorsal and columellar line present a more pleasing contour Note the change of expression about the eyes and the mouth which is a general *improvement in all these individuals after correction*

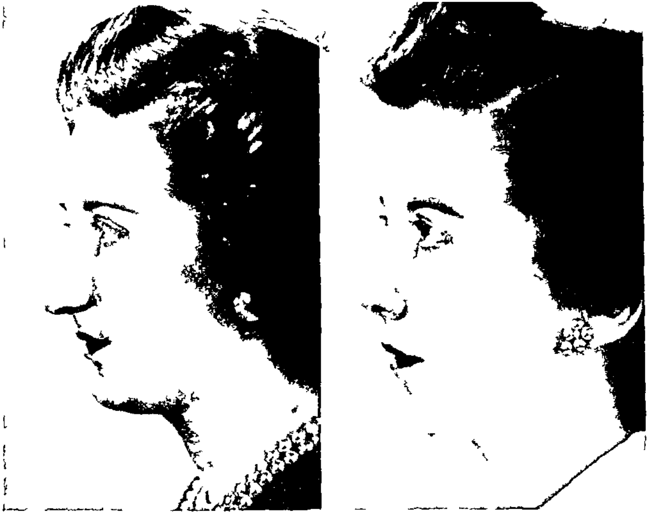

Fig 8A B Congenital high dorsal nose on a very short girl is especially conspicuous The usual procedure followed with removal of the naso labial web which *increased* the length of the upper lip In these tiny individuals a somewhat greater nasal tilt is indicated to compensate for her lack of height which will then harmonize with her facial features and stature

With this mixture the infiltration anesthetic diffuses more rapidly and does not inflate the structures nor lessen the anesthetic properties. During the past year the above mixture has reduced the number of these distortions by about 85 per cent. While there is considerably more bleeding during the operation, it is felt that if the Hyaluronidase does influence the retardation of excess fibrous and granulation tissue formation, then the added bleeding is of little consequence. Trypsin* is also being employed to reduce the edema and ecchymosis about the eyes with most favorable results. Whether these two substances have a synergistic action or not, will be reported subsequently.

After more than a quarter of a century in the field of plastic surgery, many new instruments have come to my attention, and many so-called new techniques have been advocated, which Da Costa[6] has labeled as *"an old egg taken out of cold storage and palmed off as fresh."* While a considerable number of noteworthy refinements have been developed and individual approaches have been described, the basic techniques as conceived by Joseph still remain unchanged and these include both the surgical approach and the instruments employed.

CONCLUSIONS.

The high dorsal nose is a common nasal deformity and is hereditary in nature, yet it is surprising how many will blame an injury for this familial characteristic.

As has been previously stated, in this type of deformity, the dorsal elevation of the nose must be reduced drastically in the severe types (see Figs. 5 and 6), and lesser in the mild elevations (see Figs. 7 and 8) in order to attain an appreciable result. The remaining steps to follow in this procedure are pursued as necessity demands, which the experienced operator can readily observe and continue accordingly; and by accordingly is meant to harmonize with the rest of the facial features and stature of the individual.

Any over-correction will carry the stamp of a "plastic nose," which is only too readily observed by the layman; in fact, mothers coming in for consultation with their offspring have often remarked to me, "Oh, yes, I can always tell a nose that has been corrected." A truism, indeed, when the nose has not been properly corrected; but if the nose has been properly corrected not even the most experienced plastic surgeon can detect a rhinoplasty.

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* Trypsin (Parenzyme) is being supplied by the National Drug Company, Philadelphia, Pa.

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5 Farrar, G E, Jr Medical Director, Wyeth, Inc, Philadelphia, Personal Communication

6 Da Costa, J C Modern Surgery W. B Saunders Co, Philadelphia, preface p 12, 1919

Medical Arts Building (2).

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

PROGRAM 1956

Tuesday, April 3, 1956

5:30 P.M.—*Clinical Conferences

6:30 P.M.—Dinner, Dining Room Parkland Hospital

7:30 P.M.—Program:

Parkland Hospital, Rooms 101-102
Personal Experiences in Facilitating Ophthalmic Surgery and Use of Contact Lenses in Monocular Cataracts
Dr Everett L. Goar, Houston, Texas

8:00 P.M.—External Ear Infections
Dr. E. King Gill, Corpus Christi, Texas

CLASSROOM DEMONSTRATION IN THE PHYSIOLOGY OF THE VOCAL MECHANISM.

LOUIS L. BOYARSKY, Ph.D.,

and

FRANK KODMAN, JR., Ph.D.,

Lexington, Ky.

Students taking courses in voice science, experimental phonetics, vocal pedagogy or speech pathology usually study the vocal mechanism in the form of manikins, preserved specimens or visio-audio aids (charts, films and the like). To enhance the visible display of certain anatomical and physiological relationships of the vocal mechanism the following classroom demonstration is suggested. This consists of a series of experiments carried out on an anesthetized rabbit.

PROCEDURE.

Materials: There is a working advantage in using a large rabbit. The anesthetic of choice is intravenous nembutal 0.25 milligrams per kilogram of body weight. An inexpensive electrodyne stimulator (Lab-Tronics, model N 103-B) has been found to be practical.

Musculature: After the animal shows signs of deep anesthesia, it is placed in a dorsal recumbent position. A long midline incision is made exposing the pectoral girdle. The pectorales are stimulated unilaterally with 5-10 volts, AC, 75 cps. As a point of interest, the movements of the forelimbs are observed and the pectoral origins and insertions distinguished. The electrical stimulus may be varied until maximum contraction occurs.

The sternohyoid muscle is then stimulated and its action noted. Movements of the head may also occur. To expose the trachea and larynx, the sternohyoid is divided in the midline. The student can see the movement and elasticity of the trachea during inspiration and expiration.

Editor's Note: This ms. received in The Laryngocope Office and accepted for publication, January 9, 1956.

The pectorales are next gently stripped from their sternal attachments so that the external intercostals may be viewed. Upon stimulating these muscles the local rostral movement of a single rib can be seen.

Next, attention is centered directly to the laryngeal area. The thyrohyoid can be stimulated unilaterally. The action of the cricothyroideus also occurs with direct stimulation of the muscle. At this point, the instructor may wish to elaborate in detail on the role of the individual laryngeal muscles during phonation in the human.

Laryngeal Nerve: A branch of the inferior laryngeal nerve running alongside the trachea is then gently separated from it and stimulated so that contraction occurs unilaterally. Care should be taken to avoid the large vein which runs parallel with the trachea. Exposing the nerve requires some practice on the part of the instructor.

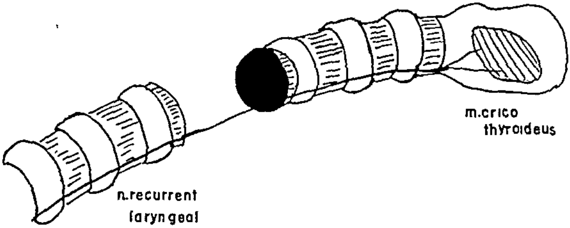

Fig. 1. Bisected Trachea Anterior View.

Vocal Folds: The trachea is severed midway between the cricoid and the sternum. Again, the branches of the inferior laryngeal nerve are avoided. The upper portion of the severed trachea is elevated and illuminated from the side. The movements of the vocal folds are readily visualized by looking into the trachea as one would a tube (see Fig. 1).

The glottis does not close completely. It enlarges during the inspiratory movement of the thorax. The glottal movements are a dramatic sight and occur despite the fact that the trachea has been cut. The dynamic relationship between glottal movements and breathing are observed readily at the low respiratory rate in the anesthetized animal. This part of the demonstration is most reinforcing as a teaching device. As a climax, the instructor may sever the inferior laryngeal nerve on one side to obtain unilateral paralysis of the vocal folds.

Diaphragm: Students are usually impressed with the diaphragm, its structure and movement during inspiration and expiration. By extending the midline incision below the diaphragm, its action may be seen and also palpated digitally.

SUMMARY.

A classroom exercise for viewing some of the physiological aspects of the vocal mechanism has been presented. The method is considered to be superior to those restricted to the use of inanimate models. The instructor can extemporize as he sees fit and readily demonstrate several meaningful physiological relationships. With practice, the entire procedure can be completed in about two hours.

ANNOUNCEMENT.

The Mount Sinai Hospital, New York, in affiliation with Columbia University announces an intensive postgraduate course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty given by Irving B. Goldman, M.D., and staff, July 14, 1956, to July 28, 1956. Candidates should apply to Registrar for Postgraduate Medical Instruction, The Mount Sinai Hospital, 5th Avenue and 100th Street, New York 29, New York.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

At the request of the Sixth International Congress of Otolaryngology, the American Express Company has arranged three post-Congress tours which will begin May 11, 1957, immediately after the close of the Congress. The tours will run concurrently and each will be accompanied by an American Express Tour Escort. All three will be in Philadelphia at the time of the Sixth International Congress of Bronchoesophagology, May 12 and 13, 1957. Tours 2 and 3 will be in St. Louis at the time of the International Congress of Audiology, May 14, 15 and 16, 1957, and Tour 2 will be in Chicago at the time of the International Voice Conference, May 20, 21 and 22, 1957. Other scientific events and a full social program are planned for the major cities visited. The itineraries for the tours are as follows:

Tour 1 (Eastern U. S.): 7 days. Cost $212.00 plus $4.10 Federal Transportation Tax. Cities to be visited include Baltimore, Philadelphia, Boston and New York.

Tour 2 (Middle West): 17 days. Cost $638.00 plus $15.98 Federal Transportation Tax. Cities to be visited: Baltimore, Philadelphia, St. Louis, Iowa City, Rochester (Minn.), Chicago, Detroit, Niagara Falls, Boston, New York.

Tour 3 (West Coast): 3 weeks. Cost $838.00 plus $30 ,7 Federal Transportation Tax. Cities to be visited: Baltimore, Philadelphia, St. Louis, Grand Canyon, Los Angeles, San Francisco, Chicago, Niagara Falls, New York.

The prices given include transportation, Pullman and hotel accommodations, meals and handling of baggage. Individuals from overseas who purchase tours abroad will be exempt from payment of the Federal Transportation Tax.

For detailed information and complete itineraries, communicate with the General Secretary, Sixth International Congress of Otolaryngology, 700 No. Michigan Ave., Chicago 11, *Ill.*, U. S. A.

SCIENTIFIC PROGRAM OF THE AMERICAN OTOLOGICAL SOCIETY, INC.

The Seignory Club, Montebello, P. Q., Canada,

Friday, May 11, 1956

I. *Address of President—Introduction to a Symposium On the Utricle*..........William J. McNally, M.D.

II. *Microscopic Structure of the Utricle*..........Catherine A. Smith, Ph.D. (By invitation).

III. *The Question of Cortical Representation of the Labyrinth*..........Wilder Penfield, M.D. (By invitation).

IV. *The Importance of the Otolothic Organs in Man Based Upon a Specific Test for Utricular Function*..........Ashton Graybiel, M.D. (By invitation).

V. *(a) Postural Vertigo Due to Partial Destruction of the Vestibular Nerve;*

(b) Summary of the Symposium..........John R. Lindsay, M.D.

VI. *Autoradiographic Studies of the Intimate Structures of the Inner Ear*..........Leonard F. Belanger, M.D. (By invitation).

VII. *Analogy Between Cochlear Fluid Motion and Formation of Surf on Sloping Beaches* (Film presentation), Jurgen Tonndorf, M.D. (By invitation).

Saturday, May 12, 1956

I. *Address of the Guest of Honor—Hearing*..........Stacy R. Guild, Ph.D.

II. *Some Recent Experiments on the Neurophysiology of Hearing*..........Robert Galambos, M.D. (By invitation).

III. *The Part Played by Psycho-Galvanic Skin Resistance. Audiometry in the Testing of Hearing of Young Children*..........Page Statten, M.D. (By invitation), and D. E. S. Wishart, M.D.

IV. *Intravascular Agglutination of the Blood: A Factor in Certain Diseases and Disorders of the Ear*..........Edmund P. Fowler, M.D.

V. *The Structure of Bone and Cartilage*..........Robert A. Robinson, M.D. (By invitation).

VI. *Functional Repair of the Middle Ear in Chronic Otitis Media*..........Professor Dr. H. Wullstein, Wurzburg, Germany (By invitation).

SCIENTIFIC PROGRAM OF THE SEVENTY-SEVENTH ANNUAL MEETING, AMERICAN LARYNGOLOGICAL ASSOCIATION.

The Seignory Club, Montebello, P. Q., Canada,

May 13th and 14th, 1956

Sunday, May 13th

A. M.

Introduction Guest of Honor . . George M. Coates, M.D.

Remarks by the President Bernard J. McMahon, M.D.

I. *"Humidity as Problem in Air Conditioning"* Arthur W. Proetz, M.D. Discussor, Lawrence R. Boies, M.D.

II. *"Histamine Releasing Drugs"* . F. C. McIntosh, M.D. (By invitation). Discussor, Aubrey G. Rawlins, M.D.

III. *"Otolaryngologic Aspects of Hypometabolism"* Jerome A. Hilger, M.D. Discussor, Francis W. Davison, M.D.

2:00 P. M.

IV. *"Functional Diseases of the Nose"* C. Stewart Nash, M.D.

V. *"Unusual Virus Diseases in Otolaryngology"* Percy E. Ireland, M.D.

VI. *"The Virus Theory of Nasal Polyp Etiology and Its Practical Applications"* . Francis L. Weille, M.D. Discussor papers V. and VI., L. W. Morrison, M.D.

VII. *"Management of Non-Malignant Growths in the Maxillary Sinus"* . . O. E. Van Alyea, M.D. Discussor, Robert L. Goodale, M.D.

VIII. *"Experimental Treatment of Recurrent Carcinoma of the Nasopharynx With Electrodesiccation, Radioactive Cobalt and X-ray Radiation"* . Francis A. Sooy, M.D. Discussor, Harry P. Schenck, M.D.

Monday, May 14th

A. M.

IX. *"Ciliary Streaming Through the Larynx"* . Anderson C. Hilding, M.D. Discussor, George Edward Tremble, M.D.

X. *"Electrical Manifestations of Recurrent Laryngeal Nerve Function"*..................................John A. Murtagh, M.D. Discussor, C. J. Campbell, M.D. (By invitation).

XI. *"Submucosal Compartmentation of the Larynx"* (Colored Slides)..Joel J. Pressman, M.D. Discussor, Paul H. Holinger, M.D.

XII. *"Carcinoma of the Larynx"*..Fernand Montreuil, M.D. (By invitation). Discussor, Henry B. Orton, M.D.

2:00 P. M.

XIII. *"Benign Growths of the Vocal Cord; a Critical Analysis of Pre-operative Diagnosis Compared With Microscopic Diagnosis"*................................Samuel Salinger, M.D. Discussor, Francis E. LeJeune, M.D.

XIV. *"Primary Malignant Tumors of the Uvula"*..........Fred Z. Havens, M.D., Richard C. Ye, M.D. (By invitation). Discussor, LeRoy A. Schall, M.D.

XV. *"Presentation of Two Laryngeal Cases"*................John B. Erich, M.D.

SCIENTIFIC PROGRAM OF THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.,

Sheraton-Mt. Royal Hotel, Montreal, Canada,

May 15-16-17, 1956

Tuesday, May 15, 1956

(Mornings Only).

9:30

1. *Presidential Remarks*....................................Dean M. Lierle, M.D., Iowa City, Iowa.

Word of Welcome........................Mr. Vincent Massey, Governor General of Canada.

9:40

Introduction of Guest of Honor..............Harold I. Lillie, M.D., Rochester, Minn.

Scientific Session

9:50

1. *Evolution of Surgical Technique in the Treatment of Carcinoma of the Larynx* Chevalier L. Jackson, M.D., Philadelphia, Pa. (Co-author: Charles M. Norris, M.D., Philadelphia, Pa.) No discussion.

10:10

2. *Developmental and Adult Anatomy of the Auditory Ossicles in Relation to the Operation for Mobilization of the Stapes in Otosclerotic Deafness*..............Barry J. Anson, Ph.D. (Med. Sc.), Professor of Anatomy, Northwestern University Medical School, Chicago, Illinois (By invitation). Theodore H. Bast, Ph.D. (Med. Sc.), Professor of Anatomy, University of Wisconsin Medical School, Madison, Wisconsin (By invitation). No discussion.

10:35

3. *Sick People in a Troubled World*........Howard A. Rusk, M.D., New York, N. Y., Associate Editor of the New York Times, Professor and Chairman of the Department of Rehabilitation and Physical Medicine of New York University's College of Medicine (By invitation). No discussion.

11:20

15 Minute Recess

11:35

4. *A Complication of the Stapes Mobilization Operation—Motion Picture*..........................Howard P. House, M.D., Los Angeles, Calif. No discussion.

11:55

5. *Some Observations on Plasma Proteins in Relation to Allergy*..................Bram Rose, M.D., Montreal, Canada, Associate Professor of Medicine, McGill University (By invitation). No discussion.

12:25

6. *Intracranial Complications of Otogenous Origin in Children Under Two Years of Age—The Role of Antibiotics*................H. James Hara, M.D., Los Angeles, Calif.

12:45

Open Discussion.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Wm. J. McNally, 1509 Sherbrooke St., West Montreal 25, Canada.
Vice-President: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Secretary-Treasurer: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Seignory Club, Montebello, P. Q., Canada, May, 1956.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Bernard J. McMahon, 8230 Forsyth Blvd., Clayton 24, Mo.
First Vice-President: Robert L. Goodale, 330 Dartmouth St., Boston, Mass.
Second Vice-President: Paul H. Holinger, 700 North Michigan Ave., Chicago 11, Ill.
Secretary: Harry P. Schenck, 326 South 19th St., Philadelphia 3, Pa.
Treasurer: Fred W. Nixon, 1027 Rose Building, Cleveland, Ohio.
Librarian, Historian and Editor: Edwin N. Broyles, 1100 North Charles St., Baltimore, Md.
Meeting: Seignory Club, Montebello, P. Q., Canada, May, 1956.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Dean M. Lierle, Iowa City, Iowa.
President-Elect: Dr. Percy Ireland, Toronto, Canada.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Mount Royal Hotel, Montreal, Canada, May, 1956.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

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Vice-Chairman: James W. McLaurin, M.D., Baton Rouge, La.
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Representative to Scientific Exhibit: Walter Heck, M.D., San Francisco, Calif.
Section Delegate: Gordon Harkness, M.D., Davenport, Iowa.
Alternate Delegate: Dean Lierle, M.D., Iowa City, Iowa.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Daniel S. Cunning, 115 East 65th St., New York 21, N. Y.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia, Pa.
Meeting: Sheraton Mount Royal Hotel, Montreal, Canada, May 15-16, 1956 (afternoons only).

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill., October, 1956.

THE AMERICAN RHINOLOGIC SOCIETY

President Dr Ralph H Riggs 1513 Line Ave Shreveport, La
Secretary Dr James Chessen, 1829 High St, Denver, Colo
Annual Clinical Session Illinois Masonic Hospital, Chicago, Illinois, October, 1956
Annual Meeting Palmer House, Chicago Illinois October, 1956

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President Dr D M Lierle, University Hospital Iowa City, Iowa
Secretary Treasurer Dr Michael H Barone, 468 Delaware Ave, Buffalo 2, N Y
Meeting Palmer House, Chicago, Ill, October 1956

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Vice President Dr Kenneth Hinderer, 402 Medical Arts Bldg Pittsburgh Pa
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President Dr J M Tato Azcuenaga 235 Buenos Aires Argentina
Executive Secretary Dr Chevalier L Jackson, 3401 N Broad St, Philadelphia 40, Pa, U S A
General Secretary Dr C E Muñoz MacCormick P O Box 9111, Santurce 29 Puerto Rico
Meeting Fifth Pan American Congress of Oto-Rhino Laryngology and Broncho Esophagology
Time and Place April 8 12, 1956, San Juan, Puerto Rico
President Dr J H Font Medical Arts Bldg San Juan P R

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

President Dr Arthur W Proetz, Beaumont Bldg, St Louis, Mo
General Secretary Dr Paul Holinger, 700 No Michigan Ave Chicago (11), Ill
Meeting Statler Hotel Washington, D C, May 5 - 10, 1957

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Vice-President Dr Jack Allan Weiss, 109 No Wabash Ave Chicago 3 Ill
Secretary-Treasurer Dr Stanton A Friedberg 122 So Michigan Ave Chicago, Ill
Meeting First Monday of each Month, October through May

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Secretary: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Meeting: Palmer House, Chicago, Ill., October, 1956.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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Vice-Chairman: Dr. Russell Page.
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Treasurer: Dr. Edward M. O'Brien.
Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

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Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building. Richmond, Va.
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Meeting: George Vanderbilt Hotel, Asheville, N. C., Sept. 16-19, 1956.

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Meeting jointly with the North Carolina Eye, Ear, Nose and Throat Society, George Vanderbilt Hotel, Asheville, N. Car., Sept. 17-18-19, 1956.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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Meeting: Quarterly, at Seven Seas Restaurant, February, May, October, and December.

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Chairman of Otolaryngology Section: Harold Boyd, M.D.

Secretary of Otolaryngology Section: Howard G. Gottschalk, M.D.

Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles, California.

Time: 6:00 P.M., first Monday of each month from September to June inclusive—Otolaryngology Section. 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Lester T. Jones, M.D., 624 Medical Arts Bldg., Portland (5), Oregon.

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Meeting: April 15-19, 1956.

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CANADIAN OTOLARYNGOLOGICAL SOCIETY
SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President: Dr. G. S. Gorrell, 110 Medical Arts Bldg., Calgary, Alta.
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Meeting: June 6-7, 1956, Chateau Frontenac, Quebec, Canada.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

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Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting:

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

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Secretary:
Meeting: Lima, Peru, 1957.

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Tesorero: Dr. Alfredo M. Petit.
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Secretary of Exterior: Dr. Aldo G. Remorino.
Secretary Treasury: Prof. Dr. Antonio Carrascosa.
Pro-Secretary of the Interior: Prof. Dr. Carlos P. Mercandino.
Pro-Secretary of the Exterior: Prof. Dr. Jaime A. del Sel.
Pro-Secretary of the Treasury: Dr. Jorge Zubizarreta.

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Secretario: Dr. S. A. Aranz.
Pro-Secretario: Dr. J. M. Tato.
Tesorero: Dr. F. Games.
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PRESENT STATUS OF STAPEDOLYSIS (STAPES MOBILIZATION).*†‡

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It is difficult to assess the present status of the revived stapes mobilization (stapedolysis) procedure for otosclerosis. Barely two years have passed since Rosen[1] re-awakened our interest in the direct approach to the ankylosed stapes footplate. Since then otologists have begun to reconsider the approach which first advocated by Kessel[2] in 1876, became well *advanced by 1890, when Miot[3] published some astute observations* on the subject.

In the past year much heat has been generated, but little light shed on this otologic chapter. The earlier failures in the stapes approach, and recent refinements and successes in fenestration surgery, obviously created a skeptical attitude toward this revival.

A presentation of my present views on surgical physiology, selection of candidates and technique will be followed by an analysis of results in 189 cases.

I. SURGICAL PHYSIOLOGY.

Basic acoustic and anatomico-pathological differences exist between fenestration and stapedolysis. Since the fenestration operation *is a detour procedure, and one in which the approach* to the perilymphatic vestibule is remote from the otosclerotic

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focus, it may be regarded as a precise procedure for a specific purpose: namely, the rerouting of acoustic energy across a new air-perilymph junction to take the place of the closed oval window footplate region. The stapedolysis approach, however, directly attacks the very pathologic lesion which is responsible for the interruption of sound transmission across the natural oval window air-perilymph junction. Since the otosclerotic disease process may occur in many varieties and in many quantitative degrees at this junction, no one operative procedure can be standardized and used in all cases in the same manner as the fenestration operation. This makes necessary a different conception of surgical attack for the direct or stapedolysis approach.

In the treatment of otosclerosis, the fenestration operation has a far more limited audiologic application than the stapedolysis approach. A physiologic deficit must occur in fenestration surgery, due to the almost complete loss of the impedance matching mechanism of the middle ear; therefore, the postoperative air conduction will seldom be better than 15 db below the preoperative bone conduction level. Since an average of 30 db loss in the speech frequencies is the lowest limit adequate for restoration of practical unaided hearing, it is necessary to start out with a good bone conduction level, one not lower than 15 to 20 db in the speech frequencies.

In stapedolysis the surgical objective is lysis of obstruction in the stapediovestibular junction so that remobilization of the footplate to air-borne sound may occur. Under ideal conditions, this approach completely respects the integrity of the tympanic membrane-ossicular chain mechanism so that the mechanical advantage of this impedance matching mechanism is not lost.

Upon achievement of maximum lysis surgically, it is possible to expect an almost complete eradication of the conductive block and an elimination of the bone-air gap, so that hearing postoperatively approaches the preoperative bone conduction level. In some cases this level will even be surpassed due to improved perilymph mobility.

Even though there is no minimal physiological deficit in stapedolysis comparable to that in fenestration, it must be recognized that deficits do occur. These deficits are either of pathologic origin or due to surgical complications.

II. SELECTION OF CANDIDATES.

Because of the above physiologic differences, one can expect greater flexibility in the application of stapedolysis, clinically. Thus, the patient with a 30 db bone conduction level and a 70 db air conduction level may still achieve a postoperative 30 db air conduction level (and practical unaided hearing) if there is no pathologic reason to interfere with "success". Such a result is usually not possible in the fenestration approach which, if successful, would not promise better than a 45 db level.

Another difference exists in selection of candidates. In fenestration surgery we were confined to the limited objective of the 30 db level for practical unaided hearing, because the surgical magnitude of the procedure made it undesirable to consider it in the majority of poor candidates where it could not possibly promise this 30 db objective.

In marked contrast, however, the lesser complexity of the stapedolysis procedure (insofar as the patient is concerned) and the lack of a minimal physiological deficit, make it advisable to consider surgical intervention even where it is impossible to expect the 30 db level. Thus the patient with 45 db bone conduction and 85 db air conduction who experiences discomfort, even with maximum output of a hearing aid, can look forward to a great improvement in his hearing status if a successful stapedolysis procedure brings his air conduction threshold back to the 45 db level. Under these circumstances (following a surgical procedure requiring one day of hospitalization and very little postoperative care) this patient can not only utilize amplification with better results but can also get along without amplification in certain circumstances which would previously have been impossible. Such a patient could have hoped at best for a 55 db or 60 db air conduction level following a successful fenestration operation.

In general, therefore, in the present early status of stapedolysis, one might consider as a candidate any patient with otosclerosis who has no specific anatomic or pathologic contraindications, and who has a bone conduction level of not lower than 45 db with a significant bone-air gap in the speech frequencies.

III. STAPEDOLYSIS—BASIC PRINCIPLES.

Since the surgical attack in stapedolysis is direct, and since the otosclerotic footplate lesions are various in degree and character, it is obvious that multiple maneuvers must be available to cope with the variations in the disease process. It is now well known that it is possible to apply a steady or intermittent force to the stapes neck and frequently achieve a very *satisfactory mobilization by a surgically induced fracture of* the footplate bony lesion, allowing restoration of function; but in this ungraded and unguided application of force it is also quite possible to dislocate the incudo-stapedial joint permanently, and to fracture one or both crura if the footplate fixation is of major degree. This "all or none" method of surgical attack does not require audiometric guidance but relies upon digital and visual monitoring of the force application.

The ultimate physiological objective in stapedolysis depends upon two factors: *a.* satisfactory lysis of the footplate obstruction, and *b.* maintenance of the functioning middle ear mechanism. It is a poor victory indeed to obtain complete footplate lysis and at the same time lose the middle ear mechanism impedance matching advantage.

In order to apply force to the footplate region adequate to produce lysis and yet insufficient to disarticulate the incus from the stapes or break the stapedial crura, it is necessary to have some method of "titration" so that the application of force can be controlled. It is obvious, of course, that such titration must be audiometric, since visual or digital impressions are unreliable in the relatively blind application of force to a small lesion which actually is primarily located on the vestibular aspect of the lateral labyrinthine wall.

The surgical audiometric technique which has been described previously by Goodhill[11] depends upon four basic steps, which may be summarized as follows:

Step No. 1—Middle Ear Closed.

This first step is performed following the completion of the flap elevation and the enucleation of the tympanic fibrous annulus from the tympanic sulcus so that the middle ear has been opened and then temporarily closed again with the skin-periosteal-tympanic membrane flap approximated to the original incision. The necessity of deferring this step until the middle ear has been opened and then again closed is to obtain a realistic audiometric graph of the traumatized tympanic membrane, annular flap, and slightly disturbed incudo-malleolar mass (as a result of the manipulation). To do this step prior to the incision and elevation would not give a true measurement for comparison with Step No. 4.

Step No. 2—Middle Ear Open.

The second step is made immediately following the first step by simply reopening the middle ear by rolling the annular flap and posterior half of the tympanic membrane anteriorly, exposing the posterior half of the middle ear with its usual landmarks. This is the step which will be used for comparison with critical Step No. 3.

Step No. 3.

This step is performed upon completion of an attempted lysis maneuver through the incudo-stapedial mass. This may involve one or more maneuvers, but it is done at a time when, in the judgment of the surgeon, sufficient stapedial motion has been produced to indicate some change. This judgment can be acquired only by experience. Subjective responses of the patient may also be used as an index.

This Step No. 3 will be compared directly with Step No. 2 for evidence of threshold shift. This is the critical point of the whole surgical procedure and may require several repetitions according to the guidance obtained by the *nomographic technique*[12] to be described. The amount of improvement which can be considered significant depends on several factors, and no set number of decibels can be considered a minimum desideratum.

Step No. 4.

Step No. 4 is performed when the posterior skin-tympanic membrane flap is replaced, as in Step No. 1, and a comparison is made between these two steps. Here again the guidance in desired degree of threshold shift will be obtained by means of nomographic information.

Because the bone-air gap is highly variable in individual cases, it is obvious that no set decibel threshold shift can be utilized as a guide, but some ratio must be available for predictive reasoning during the surgical procedure. Recently, therefore, a number of recent changes in surgical audiometry were introduced. To obtain an average threshold amplitude for comparative purposes, the "equivalent speech reception threshold" advocated by Fletcher[4] is used. This formula selects the two best responses of the 500, 1000, and 2000 cycle thresholds and averages these responses. This "equivalent SRT" is the single "figure of merit" for easy comparison of the different surgical steps. We have found the Fletcher equivalent SRT quite comparable with SRTs obtained from conventional spondee word lists.

In order to obtain a reasonably reliable method of surgical guidance, a surgical audiometric nomograph,* utilizing the Fletcher formula, has been devised. This nomograph uses the single SRT figure for bone conduction, for preoperative air conduction, for each surgical audiometric step and for postoperative air conduction. The nomograph is constructed as illustrated in Fig. 1. The ordinates (vertical axis) of the graph are in the familiar decibel ratio with respect to the normal audiometric zero level. The abscissa (horizontal axis) divisions are made equal to the ordinate, even though they represent only the discrete steps in audiometry. The preoperative bone conduction is shown by a dashed horizontal line at the proper ordinate value, using the one equivalent SRT figure of merit. This establishes the surgical objective, or the expected air conduction level, if the operation is to be an unqualified success by one of our proposed standards. The abscissae consist basically of six positions, which are used to indicate—first, the preoperative A. C. level; then the four surgical audio-

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* Nomograph, a graphic law (nomos-law).

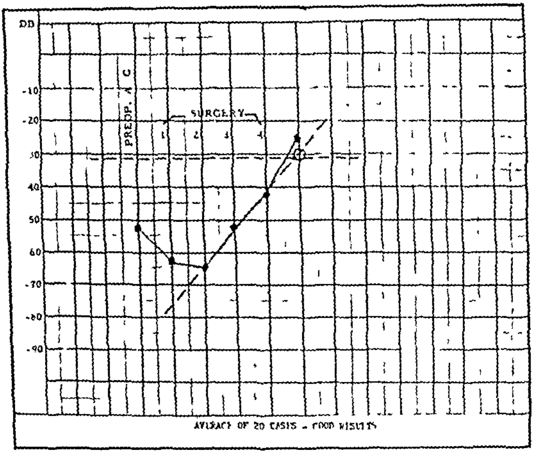

Fig 1 Average nomographic plot of surgical audiometry in 20 successful cases

metric steps; and finally the position of postoperative A. C. level which will be charted two weeks following surgery. Where more than one test is made at Step No. 3 the results are shown on the same abscissa line labeled (a), (b), (c), etc., the last one being the significant figure. Step No. 4 may be treated in the same manner when necessary.

The crucial stage in this operative approach is Step No. 3, where the surgeon has already produced some degree of force application, and perhaps some degree of lysis of the footplate ankylosis. It is at this point that a decision must be made as to whether to terminate the procedure or continue further. Thus a minimal improvement in hearing might erroneously lead the surgeon to consider that his efforts have been adequate, and he may conclude the procedure without attaining the ideal gain which could have been expected in this particular patient. On the other hand, excessive force applications at this point without adequate audiometric control may be disastrous by either destruction of one or both crura or permanent dislocation of the incudo-stapedial joint.

The nomographic technique, therefore, makes possible the utilization of predictive reasoning at this critical Step No. 3 in order to ascertain the adequacy of the lysis force exerted. The average gain to be expected between Step No. 2 and the ideal postoperative air conduction level can be roughly divided into three parts. This is, in general, true whether the required total improvement is 15 or 45 db, or any step in between. Thus, where the coordinates are equally spaced, a straight line diagonally drawn from the intersection of the preoperative bone conduction level (and the expected postoperative air conduction level) to the No. 2 step of surgery will be divided into three equal parts by the intersection of the abscissae of surgical Steps Nos. 3 and 4; therefore, regardless of whether the bone conduction threshold is high or low, and regardless of the threshold of Step No. 2, this straight diagonal line, which can be dotted in as in Fig. 1, is an excellent indication of where surgical steps Nos. 3 and 4 should appear. Thus, following lysis maneuvers, if Step No. 3 falls near or above the line (that is, to its left) it is probable that Step No. 4 and the postoperative air conduction will follow suit, providing there are no operative or postoperative complications. The reason why the expected gains between Steps Nos. 3 and 4, and between Step No. 4 and postoperative A. C. level are roughly equal to the gain between Steps Nos. 2 and 3, is due to the fact that the efficiency of the ear drum-ossicular chain mechanism as an acoustic transducer is greatest when working into the load of a normally mobile stapes. Thus, the impedance mismatch between the drum and stapes footplate is an approximate function of the degree of fixation of the latter. The level of Step No. 4 is materially influenced by the precision with which the middle ear is closed and the drum reapproximated to its sulcus attachment. If the threshold at Step No. 4 falls on or above the straight diagonal guide line, the probability of postoperative success is great.

IV. TECHNIQUE OF STAPEDOLYSIS—PRESENT STATUS.

Several features of the stapedolysis technique used by the author remain essentially unchanged since the original description.[5] The basic steps may be summarized briefly as follows:

Preoperative medication is usually limited to light barbiturates. Skin preparation relies mainly upon the use of aqueous zephiran in the canal, not only for antisepsis but also for the purpose of increasing tympanic membrane edema for easier enucleation. The entire surgical procedure is done through an ear speculum under block anesthesia. A generous omega-shaped skin flap is made on the posterior aspect of the auditory canal and the tympanic cavity opened adequately in its posterior half. Bone is removed from the postero-superior annular quadrant by the use of a burr and curette, if necessary, in order to obtain adequate exposure of the incudo-stapedial joint. Surgical audiometry is performed as previously described, using the four steps and the nomographic technique in surgical guidance.

The actual technical maneuvers necessary for adequate lysis depend primarily upon the pathologic status of the footplate region. Since the objectives are dual, namely, liberation of footplate ankylosis and preservation of middle ear impedance matching mechanism, both must be constantly considered in every operative maneuver.

A.—STABLE INCUDO-STAPEDIAL JOINT AND RIGID CRURA.

In the presence of a stable incudo-stapedial joint, with rigid crura, as determined by palpation and visual observation, the initial force application is usually trans-incudal (see Figs. 2 and 3). The needle probe (see Fig. 4) is engaged within the periosteum of the lenticular incudal process and is used to palpate for incudo-stapedial stability and degree of footplate fixation. If force application seems feasible by this technique, interrupted delicate digital vibrato pressures are transmitted through the incudo-stapedial joint to the footplate region in a direction determined largely by resistance encountered. In most instances, it will be found that the major stiffness or fixation is at the anterior limb of the footplate. Accordingly, the direction of force is usually in a posterior direction, namely, toward the posterior limb of the footplate in order to produce a surgical fracture either through the otosclerotic bone in the capsular region or through the footplate itself, thus allowing normal resumption of footplate excursions in response to acoustic energy; furthermore, the stapedial tendon in most

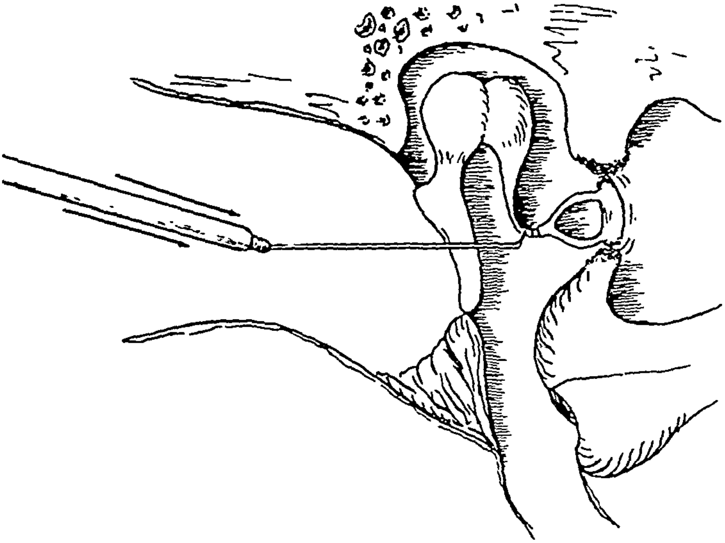

Fig. 2. Diagrammatic representation of trans-incudal stapedolysis force application.

instances will serve as a protective anchor to prevent excessive medial transmission of force with possible vestibular displacement of the fractured footplate. In some instances the direction of force will be in a superior (cephalic) direction, toward the horizontal limb of the VIIth nerve, if greatest resistance is encountered in that direction on palpation. Incudostapedial dislocation will occur more frequently when force is in an inferior direction (caudal), pulling the stapedial capitulum away from the anchored incus. There appears to be greater tolerance for slight rotation of the stapes superiorly than inferiorly. Thus, a combined maneuver in a postero-superior direction will be effective in a majority of cases.

When preliminary palpation reveals a degree of fixation too great for lysis by simple digital force, the next step is the use of the *microvibrator.*

The microvibrator consists of a dental handpiece with an *angulated "amalpak" automatic hammer attachment, to which* is added an offset right angle probe to transmit hammer-like percussion forces (see Fig. 5). The rate of motion of the vibra-

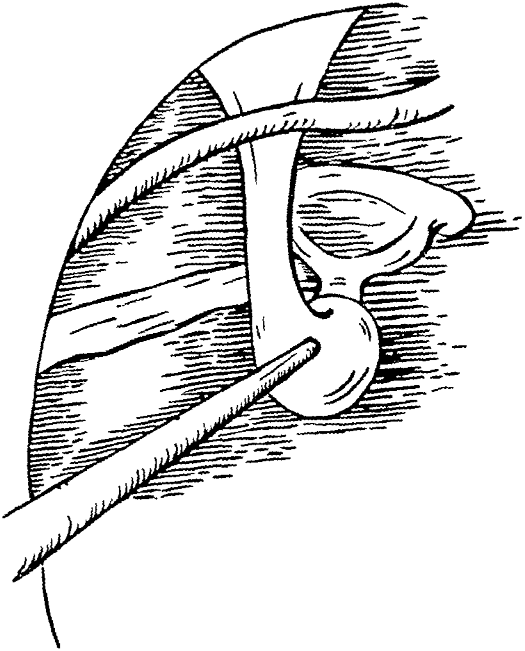

Fig 3 Trans-incudal mobilization of stapedio-vestibular joint

tor is controlled by the foot control of the dental engine in the same manner as in the use of the burr. The amplitude of motion of the microvibrator is less than 0.5 mm. Short exploratory bursts of vibration are utilized, rarely lasting longer than 0.25 second at slow speeds, and results are observed visually and monitored audiometrically. This microvibrator has a needle point and is also inserted into the lenticular process of the incus. Force is transmitted, as before, in a postero-superior direction, in short bursts. Frequently, a rigidly fixed footplate which would not respond to digital force will respond quickly to one or two short microvibrator bursts, following

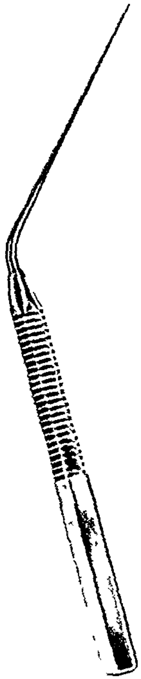

Fig. 4 Needle probe.

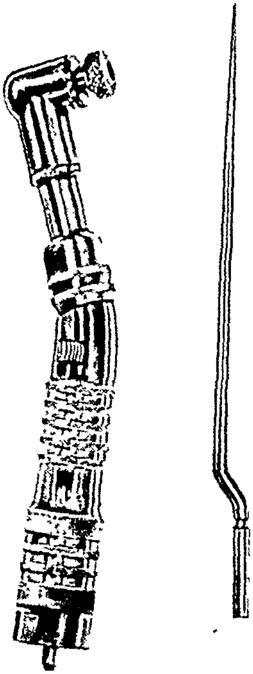

Fig. 5. Microvibrator components

which it may be possible to obtain further footplate lysis by further digital manipulation.

B.—LOOSE INCUDO-STAPEDIAL JOINT AND RIGID CRURA.

In the event that diagnostic palpation reveals a loose incudo-stapedial joint, one easily subject to dislocation, force is applied directly to the stapedial capitulum in order to prevent irreversible incudo-stapedial dislocation. A number of early attempts to cement such joints with a fast setting plastic cement "pentocryl" were only sporadically successful. Further studies to solve this problem are underway.

In such cases, the lenticular process is moved slightly away from its insertion upon the capitulum, and only sufficient capitulum area is exposed to permit application of the needle probe directly into capitular bone. When this has been successfully accomplished, gentle digital vibrato pressures are applied in the same manner as previously used in the trans-incudal approach. If such pressures are successful in producing lysis, the lenticular process is reapplied to the capitulum and the operation concluded.

If the rigidity of the footplate is so great that digital motion will be inadequate for adequate force transference, the microvibrator is substituted for the needle probe into the capitular area for direct application of microvibrator force in a posterosuperior direction. Usually, this maneuver will free even a markedly fixed footplate. Supplementary needle probe manipulation through the capitulum may be employed for further footplate lysis.

C.—ATROPHIC NON-RIGID CRURA.

In the event that palpation reveals that the crura are atrophic and elastic, or in the event that one or both crura have been surgically fractured, it is not advisable to persist in application of force either to the incus or to the capitulum. In such a situation, direct footplate lysis attempts are indicated.

D.—FOOTPLATE LYSIS TECHNIQUES.

It is difficult adequately to visualize the stapes footplate. In some cases it can be seen easily with the ordinary loupe and light, and in others with existing microscopic techniques. A significant number remains where visualization is impossible, at the present; nevertheless, surgical approach to the footplate may still be made safely, in most cases, and future improvements in stapedolysis techniques may well be based upon a *primary* application of force to the footplate region. At the present state of our experience, however, it remains the final region for lysis attempts in stapedolysis surgery. It is indicated not only in the condition where crura are atrophic, nonrigid, elastic or fractured, but also where extreme fixation or replacement has not permitted lysis by either needle probe or microvibrator through incus or through capitulum.

The simplest transmission of force to the footplate is obtained through a fine dull probe, which can be placed in the intercrural space following the curve of the anterior surface of the posterior crus. The tip of the probe will inevitably meet resistance in the footplate region and at that point gentle to and fro vibratory motion may be exerted, frequently yielding fairly prompt lysis. If such is obtained, as determined by surgical audiometry, it may still be possible to maintain impedance matching, even though the crura are elastic or partially fractured, so long as some degree of continuity exists mechanically between the tympanic membrane and the footplate region. Ideally, of course, one seeks a perfect transmission of force along the tympanic membrane-ossicular chain mechanism, but even a partial transmission under inefficient conditions is advantageous and should not be ignored.

In the event that footplate motion with the simple dull probe is ineffective, a dull microvibrator may be used in the same region, following the same placement technique and using the same type of short energy bursts, rarely lasting more than 0.25 second, and applied at a low frequency by rheostat foot control. One or two such bursts of energy application may be sufficient to produce lysis; or it may be necessary to shift the locus of application with the microvibrator in an anterior direction if posterior application fails. The footplate may have varying areas in degree of otosclerotic fixation, thus lysis may occur more readily in one region than in another; therefore, careful exploration of the footplate region is advisable before one concludes that lysis is impossible.

As a final step, if neither probe nor microvibrator force application to the footplate region is effective, gentle needle curettage of the footplate region is justified. Such curettage techniques will frequently loosen small areas of osteogenetic fixation and may allow footplate mobility which prior to that was impossible. Up to the present time, footplate curettage remains the final step in stapedolysis techniques. If there is no audiometric evidence of lysis following footplate curettage, the procedure should be concluded.

(It should be noted that successful perilymph mobilization through footplate lysis techniques occasionally produces vertigo which may last from several hours to several days, accompanied by nystagmus, nausea and vomiting. It is usually controlled well by intramuscular Dramamine, 50 mgm. every four hours.)

E.—CLOSURE.

Upon the completion of the lysis maneuvers and the demonstrations of final audiometric status, regardless of success or failure, the operative wound is finally closed. Meticulous removal of blood from the tympanic cavity is urgently indicated, and the tympanic membrane and posterior skin flap are gently replaced into their preoperative positions. The packing consists of rayon saline soaked strips to cover the skin incision, and small wedges of cellulose sponge saturated in neocortisone ointment to exert slight centrifugal pressure and keep the rayon strips in place.

F.—SURGICAL PROBLEMS.

1. Incudo-stapedial Dislocation

its tympanic membrane attachment. This, of course, seriously impairs the mechanical advantage of the middle ear mechanism, and may even create of it an obstructive lesion. It has been observed, in a few cases, that when extensive incudostapedial dislocation occurred, thresholds were better when the middle ear was open than when it was closed. It is possible in such instances that, through phase reversal, acoustic transmission was more effective via the round window, with decreased efficiency when the useless drum closed the tympanic air space and blocked the tympanic air pathways to the round window.

2. Crural Injuries.

In the application of force to the incudo-stapedial area it is inevitable that crural damage will be inflicted in some cases. Such is the case in all present techniques. It is the hope of the author that this surgical complication will eventually be eliminated completely.

The nature of crural injuries will vary with the basic pathologic status and the type and degree of force used. Such injuries may range from a simple partial greenstick fracture of one crus to complete fracture of both crura and ablation of the capitulum and crura from the footplate remnant.

Ideally, of course, crural injuries are best treated by prevention. When they occur, however, attempts should be made to replace the fragments in such positions that healing will be encouraged. Undoubtedly healing does occur in many cases, but it is not the rule probably. Observations on revisions of stapedolysis will help to clarify this point.

Frequently an injury will occur to one crus without involving the other. In such cases it is still possible to get physiologic continuity of energy transmission along the intact crus with successful impedance matching action.

(As an interesting comment, E. P. Fowler, Jr.,[*] now advocates anterior crurotomy as a preferred technique in cases limited to anterior footplate fixation. He believes that this liberation allows an effective footplate fracture and preservation of an acoustic pathway along the posterior crus which is still connected to the mobile footplate remnant.

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[*] Personal communication.

3. Hemorrhage.

Minor bleeding will frequently occur from the canal in the region of the incision, and will start either from skin or periosteal margins, or from small bone vessels. It usually subsides promptly with pressure, but it may persist. If it persists, it is a very annoying surgical problem. It may obscure the tympanic field. It may distort surgical audiometry by clotting in the oval or round window regions. If it remains as a clot it may become a precursor for tympanic fibrosis.

Bleeding should be controlled, either by pressure with epinephrine soaked cotton pledgets or by electrocoagulation of bleeding skin or periosteal vessels. The use of a polishing burr to stop small bone bleeders may be necessary.

4. Vertigo—Operative.

Momentary vertigo may be experienced by the patient during some lysis maneuvers. The patient will complain of a sense of instantaneous imbalance, which usually disappears when force is stopped. While it is not uncommon to obtain vertiginous symptoms in successful lysis, its absence is also quite common. Consequently, it is not an invaluable guide to lysis maneuvers. Apparently individual vestibular sensitivity is quite variable, and the transmission of perilymph motion from the oval window to the vestibular system is not consistently productive of vertigo. The proximity of utricle and saccule in individual cases may be related to variations in vertigo. Very rarely such operative vertigo may cause nausea and vomiting, which is very fleeting.

5. Removal of Bone Dust.

When it is necessary to remove portions of the postero-superior bony annular quadrant for better incudo-stapedial exposure, the use of a burnishing burr on a contra-angle attachment is frequently necessary. The bone dust which forms following the use of the burr should be quickly removed meticulously by the use of dry suction. This, of course, may slow up the procedure considerably, but it is preferred to the accumulation of large quantities of bone dust with the subsequent need for irrigation and massive suction. Irrigation is not dangerous, but it upsets some patients because of the caloric response.

6. *Perforations.*

It is not unusual to perforate or simply tear the tympanic membrane in the postero-inferior quadrant, particularly a very transparent atrophic tympanic membrane, during the enucleation. In such cases, the mere elevation of the skin flap-tympanic membrane segment is sufficient to produce a tear close to the annular margin in the postero-inferior quadrant. This is not a serious occurrence, since healing of the perforation almost always occurs.

In all cases, however, it is my practice to close the perforation with a small, thin, free skin graft taken from the incision margin, placed over the perforated or torn area, and covered with a rayon strip. Such a skin graft will usually take within one week with complete healing.

G.—POSTOPERATIVE CARE.

1. *Antibiotics.*

It is advisable to use prophylactic antibiotics in all cases. It is particularly necessary where a patient has been wearing a hearing aid in the operated ear. Such skin is liable to be rather edematous and of poor vitality, and external otitis, as well as otitis media may occur following surgery. If the patient has no antibiotic sensitivities, it is our practice to administer Bicillin 600,000 units intramuscularly on the morning of the operative day, and to follow this with oral tetracycline 250 mgm. t.i.d. for five days. Such antibiotic prophylaxis appears to have been effective. We have had very few postoperative infections; none of which were significant. In allergic patients, other antibiotics or sulfonamides are employed.

2. *Pain.*

There is slight pain following this surgical procedure, but it is relieved by the use of aspirin, codein or demerol.

3. Ambulation.

The patient is allowed to be ambulatory as soon as the effects of the barbiturate medication have worn off.

4. Postoperative Vertigo.

Some patients will experience immediate slight or moderate vertigo, and in some instances nystagmus may be observed. Such vertigo is usually self limited within a few hours. If it is annoying it may be alleviated by small doses of Dramamine by mouth, occasionally by injection.

5. Hearing.

The hearing level in the operated ear, which may be elevated at the time of surgery, usually drops in the postoperative period, and may remain unchanged or even lower than the preoperative level until the packing is removed.

6. Removal of Packing.

The cellulose sponge and rayon packing is usually removed about the sixth day, after preliminary application of a detergent such as phisoderm or acidolate. This allows easy removal of the packing without bleeding. If bleeding occurs, the packing is undisturbed for another few days. When the packing has been completely removed it will usually be observed that the flap is well healed, the tympanic membrane appears mobile, and in most instances is translucent. Hearing is usually up if lysis was obtained. Hemotympanum and serous effusion are encountered only rarely postoperatively and usually subside spontaneously. No further dressing is employed after the packing has been removed. It is rarely necessary to treat the canal with any type of medication.

7. Tubotympanitis.

Tubotympanitis with retraction of the tympanic membrane may occur in the early postoperative period. If this does not clear spontaneously, gentle tubal inflation by the Politzer technique or catheter is carried out. This is rarely done before the fourteenth postoperative day. It should be done very gently and carefully, with visual observation of the drum to avoid excessive pressures on the recently healed posterior skin-tympanic membrane flap.

8. Postoperative External Otitis.

Occasionally external otitis due to B. pyocyaneus or other organisms may occur. This is not uncommon in patients who have worn a hearing aid in the operated ear. The skin with poor vitality is easy prey for ordinary external environmental bacteria, and dermatitis of coccal origin may be encountered. Such external otitis is best treated by adequate cleansing with alcohol and local application of antibiotic preparations, weak acetic acid, or gentian violet.

V. POSTOPERATIVE RESULTS—AN ANALYSIS.

Since the results of stapes mobilization surgery are of very recent date, it is difficult to present a reasonably permanent evaluation at this early date. In the recent era, since Rosen[1] reported his first cases, only two years have elapsed, and the observations of this short period must be considered tentative and temporary; nevertheless, a number of deductions can be obtained from a careful analysis of immediate results. In this section immediate results will be presented first, followed by a discussion of analysis of failures, complications, reankyloses, revisions, fenestration of failures, revision of fenestrations by stapedolysis techniques, and finally the application of binaural stapedolysis. It must be emphasized again that these are very early observations and that long range studies may change the evaluations considerably in one direction or another.

The results of the author's series are based upon a study of more than 200 patients. Stable postoperative audiometric information is available on 189 cases. These 189 cases will be subjected to an analysis from varying points of view, and the results will be discussed and plotted graphically.

A.—IMMEDIATE POSTOPERATIVE RESULTS OF STAPEDOLYSIS.

1. Results of Other Observers.

A. Rosen[6] in April, 1955, reported results in 211 cases, which were divided into A, B, and C groups, depending upon preoperative bone conductor levels. Of his total number of cases, apparently 37.3 per cent were classified as successes, 29.4 per cent having reached the 30 db level and 7.9 per cent the 35 db level. His criteria for success were typically fenestration criteria.

B. Meurman and Meurman,[7] in August, 1955, giving 63 primary results, reported good improvement in all groups (A, B and C) in 39.6 per cent and slight improvements in 19.1 per cent, a total of 58.7 per cent improved.

C. Scheer[8] in September, 1955, reported results in 100 cases. The footplate was found mobile in 3 per cent, the crura were fractured in 30 per cent, mobilization, successful and unsuccessful, was achieved in 67 per cent; 33 per cent of Scheer's patients were improved to the 30 db level; 13 per cent were improved to the level of preoperative bone conduction; a total of 7 per cent minor gains and successes amounted to 40 per cent. His total failures, including no gains and fractured crura, amounted to 57 per cent, and 3 per cent of the footplates were found mobile at the time of surgery.

2. Analysis of the Author's Cases—189 Cases.

As stated above, the observations of stapedolysis are based upon the author's experience in over 200 cases. Complete immediate postoperative audiometric data are available in 189 cases. These are considered in analysis, both as a total group of 189 and as two groups of the first 100 and the second 89 cases. This somewhat arbitrary division into two groups is based upon both chronological order and technical change. The entire group of 189 cases was operated upon between November, 1954, and December, 1955. The first 100 cases were done between November, 1954, and July, 1955; and the second group of 89 cases done between July, 1955, and December, 1955.

Undoubtedly, increased experience and technical changes were responsible for some of the differences, which will be observed in the following analyses. The technical changes included *a.* a more precise technique of lysis maneuvers based upon differences in gross pathologic variations, and *b.* the use of nomographic guidance in surgical audiometric techniques.

It is perhaps unfortunate in many ways that stapes mobilization (stapedolysis) is at present closely compared with fenestration, not only in technique but also in results. It is understandable that such comparisons are necessary in view of the history of the approaches; nevertheless, the fields covered by these two surgical techniques are different, although overlapping, and the results considered satisfactory are not necessarily the same in both approaches.

In fenestration surgery, the goal has usually been what is called practical or unaided hearing. While definitions of this objective vary, it is generally conceded that an air conduction level 30 db below the zero reference level is the lowest figure that can be used without qualification. The magnitude of the fenestration operation is such that it has usually not been considered advisable in cases where the predicted result could not attain this 30 db level objective.

Stapedolysis, with a much lower order of surgical trauma, has provided satisfactory gains in air conduction even in cases where the preoperative bone conduction was so low that unaided or practical hearing was obviously improbable. Thus, success in stapedolysis may be defined either *a.* in terms of the achievement of the practical hearing level (30 db) or *b.* in terms of the removal of the conductive block as is evidenced by a postoperative air conduction which reaches or surpasses preoperative bone conduction.

Thus, in all fairness, the title of "success" in stapedolysis surgery must be a dual concept, and must include either the achievement of the 30 db level or the elimination of the conductive block, as evidenced by an eradication of the bone-air gap.

The following definitions will clarify our concepts in evaluation of results:

A. "Thresholds".

The values of acoustic thresholds are stated in terms of the *equivalent SRT* using the Fletcher[4] formula (an average of the two best threshold responses at either 500, 1000 or 2000 cycles per second). Thus, while audiometric measurements are in 5 db steps, the equivalent SRT values here used may vary in 2.5 db steps.

B. "No Change".

"No change" is used when the postoperative air conduction level is within plus or minus 5 db of preoperative air conduction.

C. "Losses".

"Losses" will be described where the last postoperative air conduction level was 7.5 db or more below preoperative air conduction.

D. "Partial Gains".

"Partial gains" will be described as cases in which the last postoperative air conduction is 7.5 db or more above the preoperative air conduction, but less than "success".

E. "Successes".

"Successes" will be defined as either *1.* cases in which the *last postoperative air conduction is above the level, at the same* level as, or not more than 7.5 db below preoperative bone conduction; or *2.* air conduction is not more than 30 db below the normal zero reference level. (Since audiometric measurements may vary by 5 db, and since our figures change in 2.5 db steps, the 7.5 db value is used as being a reasonable lower limit for "success".)

The following discussion will relate to the graphs which have been constructed in an attempt to analyze the results of stapedolysis, both as independent results and also in some cases in comparison with fenestration surgery. Thus differences and similarities between the two surgical approaches will be displayed in various types of candidates.

Fig. 6-A shows the distribution of the preoperative bone conduction levels of 189 candidates for stapedolysis. They are assembled in five db groups and are further segregated to indicate their probable classification as fenestration candidates. This histogram, which shows the number of cases in each

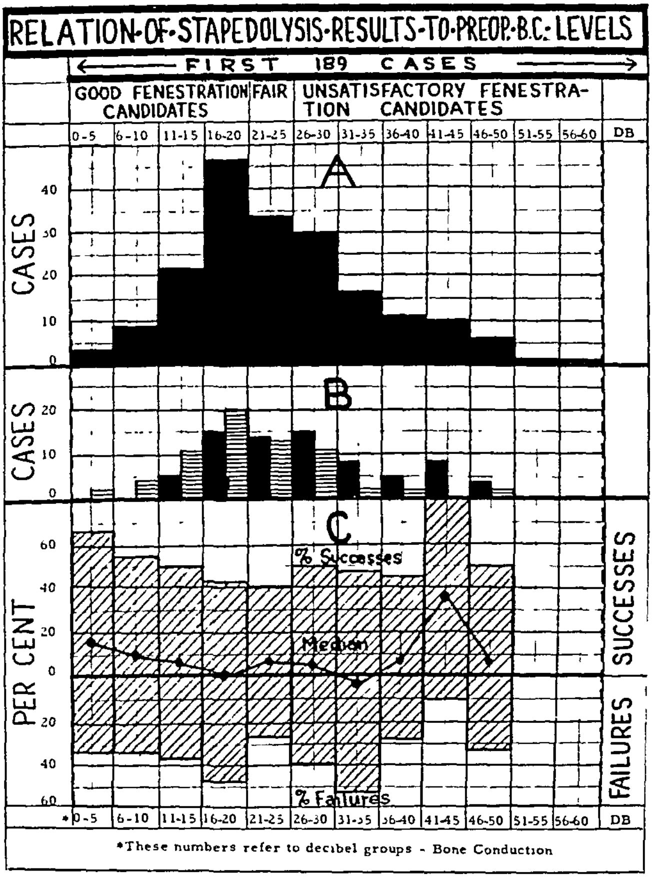


Fig. 6. Relation of stapedolysis results to preoperative bone conduction levels in 189 consecutive cases.

A—Distribution of preoperative bone conduction levels.

B—The number of cases achieving success within the various A groups. The crossbarred areas show cases within 30 db of normal air conduction. Shaded areas show cases within 7½ db of preoperative bone conduction. Areas side by side in the same group qualify as successes in both categories. The largest number of cases shown in each group is the total for that group.

C—Per cent successes are shown above the zero line. Per cent failures are shown below the zero line. The "median" line shows that results do not consistently favor any bone conduction, where there are significant numbers of samples.

group on the ordinate or vertical axis, demonstrates clearly the difference in the fields covered by the two surgical approaches. Assuming that ideal fenestration candidates are those with bone conduction not poorer than 20 db, it will be seen that there were 80 such candidates in the 189 cases shown. In the group with bone conduction between 21 and 25 db below zero, there were 34 cases which would be considered fair candidates for fenestration surgery. This leaves a group of 75 patients who would have been considered unsatisfactory candidates for fenestration; nevertheless, an analysis of the *data in Fig. 6-B indicates that in this last group of 75 patients* considerable relief for the mixed type of deafness in otosclerosis can be expected from stapedolysis. Graph (see Fig. 6-B) shows the distribution and the two types of "success" in stapedolysis. Cases which are considered successful by virtue of having reached the postoperative air conduction level of 30 db or better are shown crossbarred; those which are considered successful because the conductive block (the bone-air gap) has been essentially removed regardless of the final air conduction level are shown in solid blocks. Where the two types of successes are shown side by side in a 5 db group, they qualify as successes under both definitions. For example, in the 16 to 20 db group, the total number of successes is 20, of which 15 qualify under both headings, and five cases qualify only in the 30 db class. As might be expected, the 30 db class is predominant in the 16 to 20 db group, and higher, while at preoperative bone conduction values below 20 db the elimination of the conductive block is most productive of success. It is an interesting fact, however, that there are many 30 db levels achieved from preoperative bone conduction groups that are much below the accepted fenestration candidate standards. It will be noted that 39 successful cases were obtained in this region of unsatisfactory fenestration candidates.

In Fig. 6-C, the percentage of successes and failures are shown relating A and B in various groups to see whether useful information might be obtained for candidate selection criteria in stapedolysis. Thus, total successes in each group are plotted in percentages above the zero reference line, and all failures, that is, all cases that failed to show over a five db gain in air conduction, are plotted below the reference line; partial gains are not considered. Where the midpoint of the resultant box falls on the zero line, the chances for success or failure are even; if the midpoint is above the line, the probability favors success or vice versa. Even if the criteria for success or failure are debatable in terms of db levels, the line connecting these midpoints will show any existing trend toward success or failure in these groups. In the groups from minus 10 db to minus 35 db, where there are sufficient cases to be significant, there appears to be little real advantage in any area of preoperative bone conduction. (The rise at both ends of the curve may disappear, or may be confirmed with more data.)

In Figs. 7 and 8, the entire group is analyzed in terms of air conduction gains and losses, and they are classified in terms

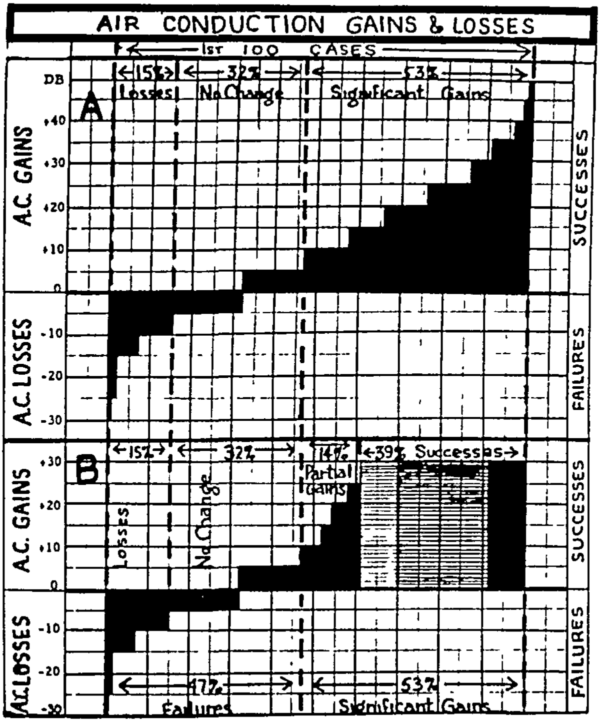


Fig. 7. Air conduction gains and losses—first 100 consecutive cases.

A—Gains and losses without respect to success.

B—Same as A except that all gains achieving success by either definition are shown as a group. White crossbarred areas are successes to 30 db level. Gray crossbarred areas are successes both to the 30 db level and in eradication of BC-AC gap. Solid areas are successes by eradication of BC-AC gap only.

of change from a zero level. Fig. 7 shows the results of the first consecutive 100 cases, and Fig. 8 shows the results in the second consecutive 89 cases.

Fig. 7 is plotted differently from the histogram (see Fig. 6), because the gains and losses to be displayed show most clearly when plotted above and below a zero reference line. Thus the db gains or losses are shown as ordinates on the vertical axis, and the number of cases in each five db group are shown on the horizontal zero axis. The first 100 cases shown in Fig. 7 may be analyzed as follows: In Fig. 7-A, the gains and losses

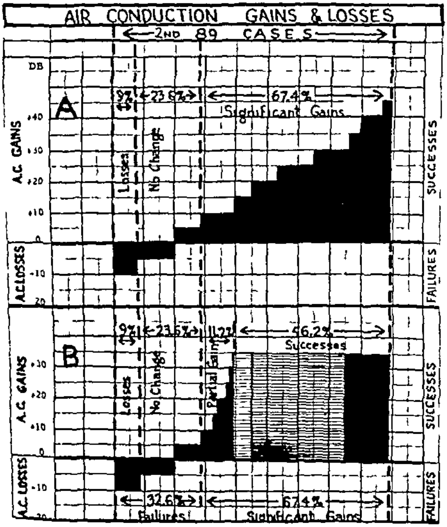


Fig 8 Air conduction gains and losses—second group of 89 consecutive cases

A—Gains and losses without respect to success (same as A in Fig 7)

B—Same as A except 'Successes' are shown as a group without regard to gain Note the percentage increase in 'Successes' over the first 100 cases shown in Fig 6-B White crossbarred areas are successes to 30 db level Gray crossbarred areas are successes both to the 30 db level and in eradication by BC-AC gap Solid areas are successes by eradication of BC-AC gap only show 15 per cent losses, 32 per cent no change, and 53 per cent with significant gains of 10 db or over.

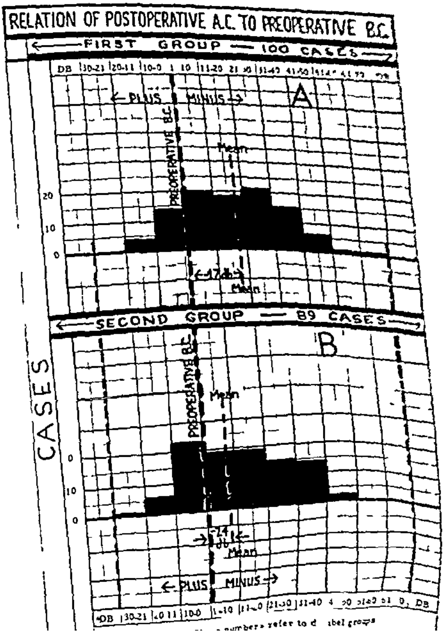
Fig. 7-B shows the same data except that the successes are shown as a group without regard to actual db gain level, but they are divided into two categories of success, as previously described in Fig. 6-B. Thus, a total of 39 per cent of the cases in the first 100 may be classified as successes by one or both criteria of success; 14 per cent showed useful gains but were below the criteria necessary for success, and 47 per cent could be classified as failures (no change or losses).

In Fig. 8, which analyzes the second consecutive group of 89 cases, it will be noted with reference to Fig. 7 that the losses encountered are less severe, with none over 10 db, and are reduced from 15 per cent to 9 per cent. The no change category has been reduced from 32 per cent to 23.6 per cent. The feature of greatest importance, however, is the increase in the percentages of successes from 39 per cent to 56.2 per cent, with partial gains of 11.2 per cent. It is of interest to note that the increase in successes is more marked in the group with bone-air gap eradication rather than in the 30-db level group. This may reflect a definite improvement in technique.

It has been proposed by Davis and Walsh[9] that fenestration surgery be evaluated by the "surgical deficit" method, whereby the postoperative air conduction is compared to an expected value. This "expected" value is the mean resultant of a large number of cases, and for convenience is referred to preoperative bone conduction. Thus, they find that the expected postoperative level for fenestration as derived from 196 consecutive cases is 20 db below preoperative bone conduction. Shambaugh,[10] reporting on 164 consecutive cases, finds an expected value of 15 db below preoperative bone conduction.

Once the expected mean has been established for any given technique, or any given surgeon, the actual individual result is compared to the expected result, and the algebraic difference in decibels is stated as a surgical deficit. This method of evaluation has the advantage that it eliminates the more or less artificial definitions of success or failure and candidate classification, and permits the direct comparison of different techniques and procedures in surgery.

This surgical deficit technique is illustrated in its [illegible] tion to stapedolysis in Figs. 9-A and B, in which the [illegible] ship of postoperative air conduction to preoperative bone conduction is compared. In Fig. 9-A, the resultant [illegible] would rotate about a point 17 db below preoperative bone conduction. In the second group of 89 cases (see Fig. 9-B), [illegible]



proved technique apparently has moved the median point up 9.5 db to about 7.5 db below preoperative bone conduction. It is, of course, hoped that surgical technique improvements will make for further advances, thus pushing the expected value further toward, or possibly even above preoperative bone conduction. It is of interest, however, that even at this early stage the expected result derived from unselected candidates is still 7.5 db better than the best presently accepted figure for fenestration of 15 db.

B.—COMPLICATIONS.

Complications of stapedolysis surgery are singularly rare. They may be divided into two groups: *a.* cases where hearing losses have occurred postoperatively, and *b.* other complications.

The first group *a.* has been described above. A number of threshold losses occurred as can be seen from a study of previous graphs; however, most of these losses were of academic interest only, since they did not materially change the social adequacy of the patient's hearing ability. No case of labyrinthitis or significant cochlear damage occurred in the author's series. All losses recorded were essentially conductive impairments. The absence of labyrinthitis and concomitant cochlear damage is in contrast to reports of some fenestration series.

The second group of miscellaneous complications is pleasantly small. No cases of facial paresis or paralysis were encountered. No cases of persistent vertigo were found. The longest postoperative duration of vertigo in one case was seven days. Two patients. among the first ten cases operated, had persistent dry central perforations. No cases of otorrhea occurred. Chorda tympani paresthesia was present temporarily in only a few patients. It has persisted in only one patient of the series. There were no surgical or post-surgical mortalities.

It may be stated in summary that the complications of stapedolysis are very few and minor.

both, was necessary in the postero-superior annular quadrant. In this group of cases no significant trends toward success or failure could be found.

Hemorrhage from the incision was encountered in 60 per cent of all cases; nevertheless, this factor did not appear to be significant in producing success or failure.

Tympanic perforations occurred in 17 per cent of all cases. Most were tiny tears, and all were immediately closed with a small skin graft as previously described. (Only two persistent perforations are present in this entire group.) In this group there was no deviation from the previous result averages.

Incudo-stapedial dislocation occurred temporarily in 9 per cent and probably permanently in 10 per cent. Here, probably because of the small number of cases, no significant trends were found.

Crural injuries were observed in 14 per cent ranging from partial to complete fractures. Here again, no significant departures from the previous data were found.

In 10 per cent of the cases which were adequately lysed or mobilized, according to surgical audiometric nomographs, postoperative success did not occur. In the absence of significant operative deviations it must be concluded that these represented rather *immediate re-ankyloses.* This group is, of course, classified above under failures.

In the final analysis, except for the 10 per cent of immediate re-ankyloses, it seems that *simple lysis failure* is the main cause of "immediate operative failures". Review of operative nomographs shows a high degree of correlation of nomographic failures and failure to mobilize.

D.—RE-ANKYLOSIS.

Evidence of late partial or complete re-ankylosis of the footplate with threshold losses in primary successful cases has been found in 11 cases in this series of 200; however, this is undoubtedly a low figure and a premature survey, since the series of 200 cases extends from November, 1954, to December, 1955. The ultimate ratio of incidence of re-ankylosis is an exceedingly important figure in any analysis of this surgical approach.

As Rosen[6] pointed out, one wonders why all of the successful cases do not re-ankylose; yet, in the few reports so far, very few have been reported. It is possible that capsular bone, especially pathological capsular bone, does not have good regenerative powers; neverthelss, the fact that, in little over a year, 11 cases have shown significant threshold losses, is cause for serious study.

A preliminary survey of these 11 cases shows partial re-ankylosis in six and probably complete re-ankylosis in five cases, as determined by audiometric studies. No special differences as to age or sex are apparent. Duration of disease prior to surgery does not appear to be a significant factor. One very important fact is that of this group of 11 cases, only one patient had preoperative bone conduction better than 20 db. The rest were definitely poor candidates with preoperative bone conduction levels ranging from 35 db to 55 db. Surgical re-exploration is planned for most of these cases, prior to fenestration. None has been explored yet.

E.—STAPEDOLYSIS REVISION OF STAPEDOLYSIS FAILURES.

Three cases in which stapedolysis attempts were primary failures were revised by the stapedolysis technique. In two cases, lysis was obtained with improvement in hearing, probably due to more effective application of force in a previously partly mobilized footplate. In the third case, failure to mobilize again was experienced.

F.—FENESTRATION OF STAPEDOL

Three fenestration operation
author in stapedolysis failures.
tive results have been obtained
servations are unavailable, of c

No special surgical problems were encountered in these cases. The preparation of the tympanomeatal membrane was unusually easy, probably due to previous elevation in stapedolysis. It should be noted that the stapedolysis incision of the author is intentionally generous, in order to avoid a scar in the region of the thin portion of the membrane which would be used to cover the fenestra.

G.—STAPEDOLYSIS REVISION OF PREVIOUS FENESTRATION FAILURES.

Prompted by the Rosen[6] case report in which he successfully mobilized the stapes in a post fenestration closure, the author attempted this procedure in five cases. In none of these cases was a hearing gain obtained, even though visual evidence of mobilization appeared probable in at least four of these cases.

It is possible that this problem may yet lend itself to solution. If the footplate ankylosis can be adequately lysed, hearing gains should be possible if a successful myringo-stapediopexy could be obtained. Under other circumstances, hearing gains might be obtained through a phase reversal phenomenon, by a permanent tympanic perforation, in which the round window would become the entrance route of air-borne sound to the perilymph space.

H.—BINAURAL STAPEDOLYSIS.

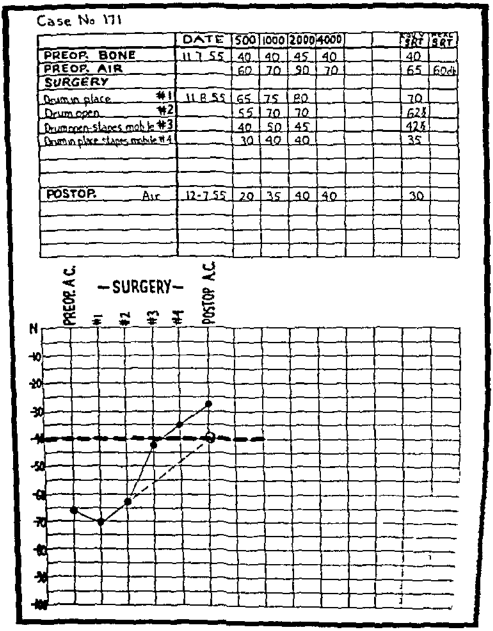
In the 200 ears reported above, 34 represented the second ear in as many patients (total 68 ears). In seven of these patients (14 ears) stapedolysis failures were encountered binaurally. In 11 cases (22 ears) successes were obtained binaurally. In 16 cases (32 ears) successful results were obtained on one ear (16), even though failures were obtained on the other ear (16).

I.—EXAMPLES OF SUCCESSFUL AND UNSUCCESSFUL RESULTS.

Specific examples of both successful and unsuccessful cases may be of interest.

1. *Case No. 171 (see Fig. 11).*

51-year-old female. This patient with poor bone conduction has been restored to unaided hearing.



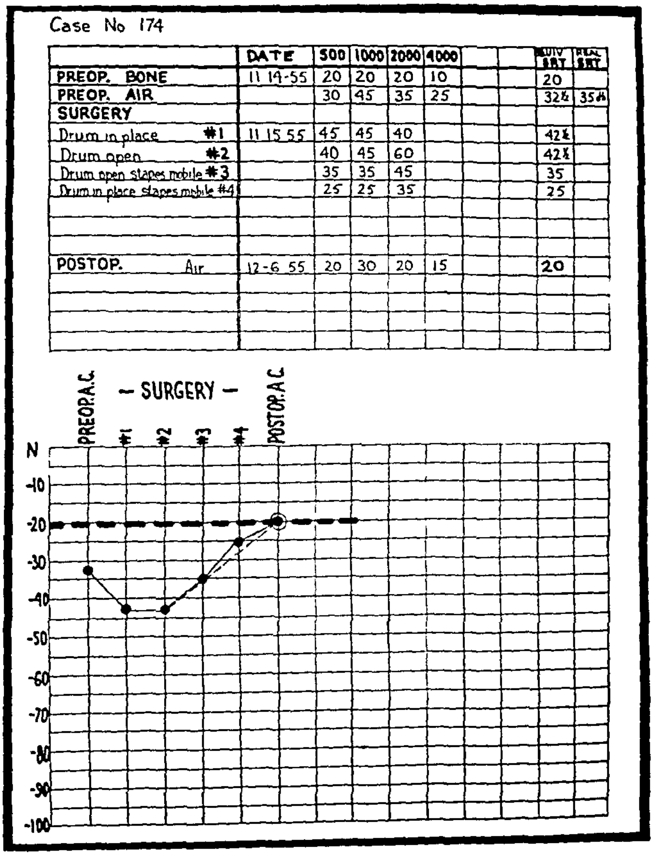
Case No 171

	DATE	500	1000	2000	4000			SRT	SRT
PREOP. BONE	11 7 55	40	40	45	40			40	
PREOP. AIR		60	70	90	70			65	60db
SURGERY									
Drum in place #1	11 8 55	65	75	80				70	
Drum open #2		55	70	70				62½	
Drum open-stapes mobile #3		40	50	45				42½	
Drum in place stapes mobile #4		30	40	40				35	
POSTOP. Air	12-7 55	20	35	40	40			30	

Fig. 11.

2. Case No. 174 (see Fig. 12).

33-year-old female. This patient with good bone conduction has reached her bone conduction level.



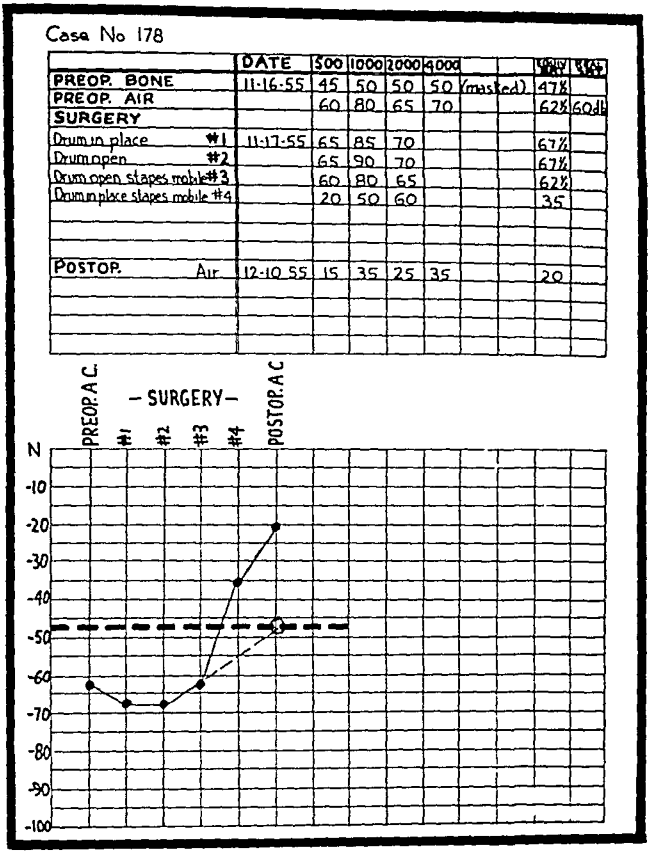
Case No 174

	DATE	500	1000	2000	4000			EQUIV SRT	REAL SRT
PREOP. BONE	11 19-55	20	20	20	10			20	
PREOP. AIR		30	45	35	25			32½	35½
SURGERY									
Drum in place #1	11 15 55	45	45	40				42½	
Drum open #2		40	45	60				42½	
Drum open stapes mobile #3		35	35	45				35	
Drum in place stapes mobile #4		25	25	35				25	
POSTOP. Air	12-6 55	20	30	20	15			20	

Fig. 12.

4. Case No. 178 (see Fig. 14).

60-year-old female. This patient with preoperative B.C. averaging 47.5 db reached a postoperative A.C. level of 20 db.



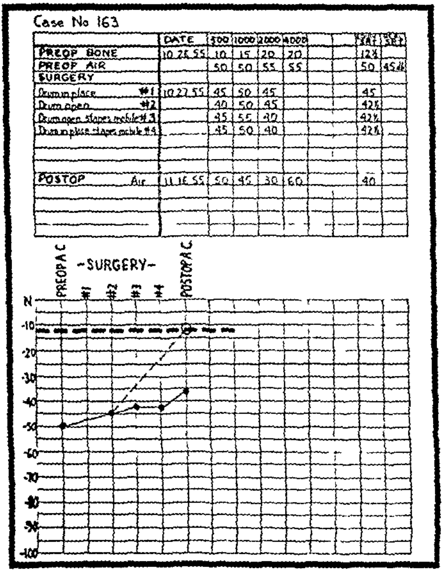
Case No 178

	DATE	500	1000	2000	4000			[illegible]	[illegible]
PREOP. BONE	11-16-55	45	50	50	50	(masked)		47%	
PREOP. AIR		60	80	65	70			62%	60db
SURGERY									
Drum in place #1	11-17-55	65	85	70				67%	
Drum open #2		65	90	70				67%	
Drum open stapes mobile #3		60	80	65				62%	
Drum in place stapes mobile #4		20	50	60				35	
POSTOP. Air	12-10 55	15	35	25	35			20	

Fig. 14.

5. Case No. 163 (see Fig. 15).

31-year-old female. This patient with excellent preoperative B.C., and a good fenestration candidate, nevertheless failed to reach a satisfactory level.



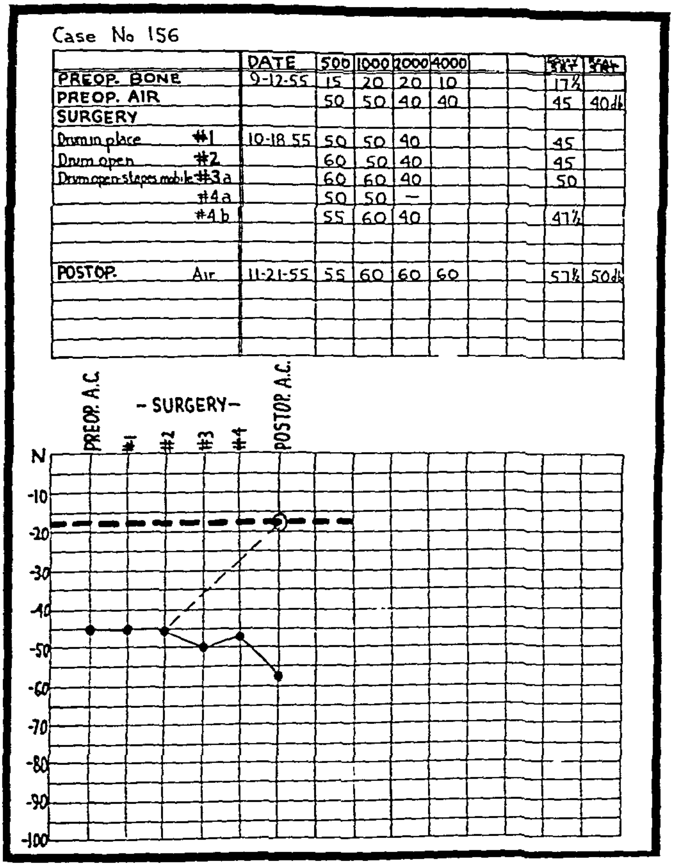
Case No 163

	DATE	500	1000	2000	4000			[illegible]	[illegible]
PREOP BONE	10 28 55	10	15	20	20			12½	
PREOP AIR		50	50	55	55			50	45db
SURGERY									
Drum in place #1	10 27 55	45	50	45				45	
Drum open #2		40	50	45				42½	
Drum open, stapes mobile #3		45	55	40				42½	
Drum in place stapes mobile #4		45	50	40				42½	
POSTOP Air	11 16 55	50	45	30	60			40	

Fig. 15.

6. Case No. 156 (see Fig. 16).

24-year-old male. This patient with excellent preoperative B.C. failed to reach a satisfactory level.



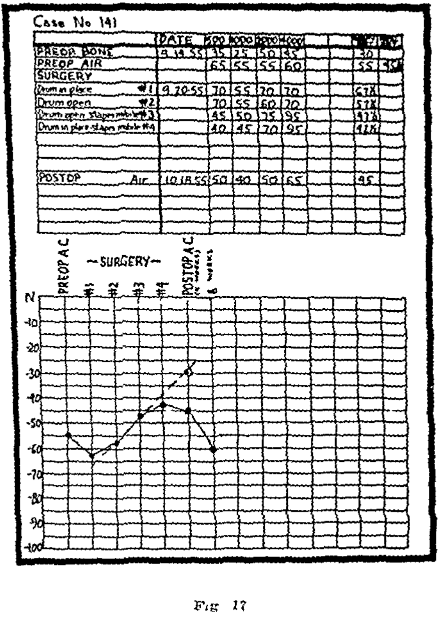
Case No 156

	DATE	500	1000	2000	4000			[illegible]	[illegible]
PREOP. BONE	9-12-55	15	20	20	10			17½	
PREOP. AIR		50	50	40	40			45	40db
SURGERY									
Drum in place #1	10-18 55	50	50	40				45	
Drum open #2		60	50	40				45	
Drum open-stapes mobile #3a		60	60	40				50	
#4a		50	50	–					
#4b		55	60	40				47½	
POSTOP. Air	11-21-55	55	60	60	60			57½	50db

Fig. 16.

7. Case No. 141 (see Fig. 17).

59-year-old female. This patient with poor preoperative B.C. nevertheless showed excellent nomographic surgical progress; however, her two-week postoperative threshold fell below expectations, and an eight-week postoperative threshold showed complete re-ankylosis.



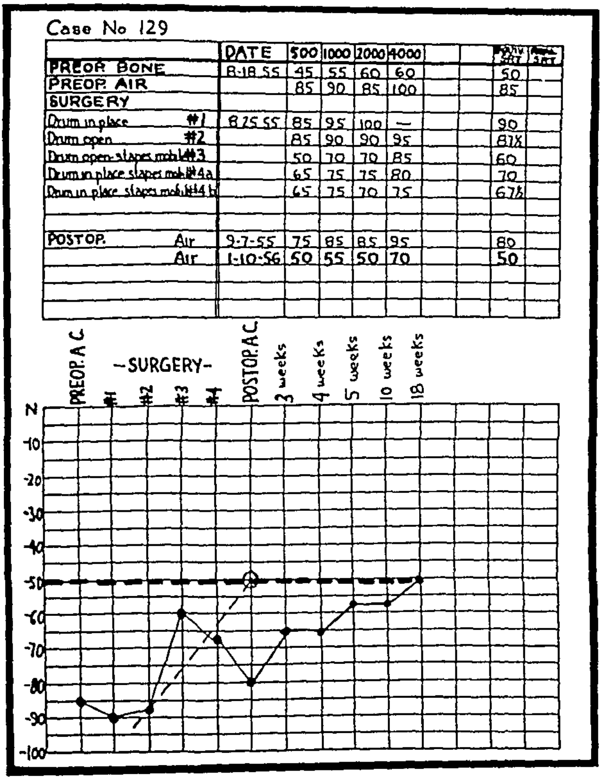
Case No 141

	DATE	500	1000	2000	4000			[illegible]	[illegible]
PREOP BONE	9.14.55	35	25	50	35			30	
PREOP AIR		65	55	55	60			55	55%
SURGERY									
Drum in place #1	9.20.55	70	55	70	70			67%	
Drum open #2		70	55	60	70			57%	
Drum open stapes mobile #3		45	50	75	95			41%	
Drum in place-stapes mobile #4		40	45	70	95			41%	
POSTOP Air	10.18.55	50	40	50	65			45	

Fig 17

8. Case No. 129 (see Fig. 18).

55-year-old male. This patient with poor preoperative B.C. showed excellent nomographic progression surgically. Unusual hemorrhage complicated the procedure with marked early postoperative regression; nevertheless, slow recovery occurred with subsidence of the hemotympanum, and 18 weeks postoperatively the preoperative B.C. level was reached. A gain of 35 db.



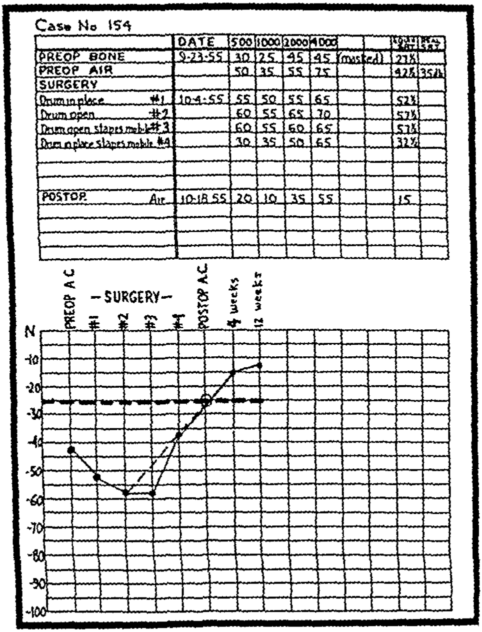
Case No 129

	DATE	500	1000	2000	4000			Av SRT	[illegible]
PREOP. BONE	8-18-55	45	55	60	60			50	
PREOP. AIR		85	90	85	100			85	
SURGERY									
Drum in place #1	8-25-55	85	95	100	—			90	
Drum open #2		85	90	90	95			87½	
Drum open-stapes mobil #3		50	70	70	85			60	
Drum in place stapes mobil #4a		65	75	75	80			70	
Drum in place stapes mobil #4 b		65	75	70	75			67½	
POSTOP. Air	9-7-55	75	85	85	95			80	
Air	1-10-56	50	55	50	70			50	

Fig. 18.

9. Case No. 154 (see Fig. 19).

28-year-old male. This case was characterized by temporary incudo-stapedial dislocation. Repositioning of the bones was successful, and subsequent nomographic course shows an excellent result.



Case No 154

	DATE	500	1000	2000	4000			[illegible]	[illegible]
PREOP BONE	9-23-55	30	25	45	45	(masked)		27%	
PREOP AIR		50	35	55	75			42%	35/L
SURGERY									
Drum in place #1	10-4-55	55	50	55	65			52%	
Drum open #2		60	55	65	70			57%	
Drum open stapes mobile #3		60	55	60	65			57%	
Drum in place stapes mobile #4		30	35	50	65			32%	
POSTOP. Air	10-18 55	20	10	35	55			15	

Fig 19.

Thus it can be readily seen that there are many apparent inconsistencies in these results and much study is necessary for modifications in technique.

J.—TINNITUS.

An evaluation of tinnitus is in progress at the present. To date, it seems that tinnitus has been relieved in most successful cases and unchanged in failures. There are exceptions, however. Several successful cases audiometrically, report increased tinnitus. Several failures report tinnitus disappearance. Several failures report tinnitus increases.

VI. CANDIDATE SELECTION TRENDS.

So far no method of real value has been devised for the selection of candidates in stapedolysis. A number of possible criteria have been considered in the approach to a selection formula; namely *1.* preoperative bone conduction; *2.* preoperative air conduction; *3.* preoperative air-bone gap; *4.* age of candidate; and *5.* duration of disease. Duration of disease, the last item listed, has not been studied in this series because of difficulty in obtaining meaningful figures from the average patient. It is not too difficult to determine the age at which the deafness became a social and economic problem, but it is very difficult to determine the actual onset of hearing deterioration. Consequently, this factor has not been evaluated seriously in our study at the present time.

Bone conduction has been the base for the selection of fenestration candidates, and thus there has been some tendency to continue selection by the same formula in stapedolysis. We have already examined the influence of various bone conduction levels, and the probability of success, in Fig. 6-C, and found that if we limit our consideration to those groups of the 189 total that provide over ten samples (–10 to –40 db) we do not find a decisive trend toward success or failure over this area. Too few cases are involved at levels above –10 or below –40 db to consider the rise shown at both ends of the median line as typical. Thus from the data at hand, bone conduction alone does not seem to contain preoperative information of value in candidate selection for stapedolysis.

Air conduction levels would seem logical as one approach to the degree of fixation, and this factor is explored in Fig. 20. Preoperative distribution of air conduction levels, for the 189 cases as shown in Fig. 20-A. In Fig. 20-B, the percentages of success and failure are treated as in Fig. 6-C, to obtain the same type of graph. The median line displays a very slight downward trend with increased air conduction loss; however, there is nothing here that would be sufficiently significant to set up rigid selection criteria.

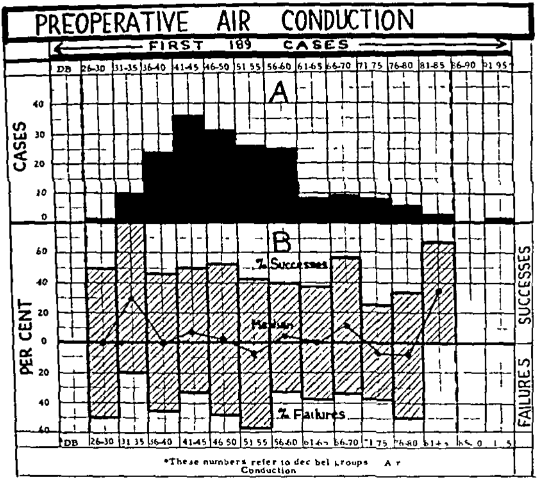


Fig. 20 Preoperative air conduction levels in 189 consecutive cases

A—Distribution of air conduction levels The extreme case shown at 91-95 db made a significant gain but was not a success.

B—Percentage of successes in each 5 db group is plotted below the same line The "median" line derived from both appears to be essentially random in character, where the number of samples are significant

The question of bone-air gap as a method of exploring selection criteria is studied in Fig. 21. The distribution of the bone-air gap is shown in Fig. 21-A, while the percentage of successes and failures are plotted as before in Fig. 21-B. Here, again, there is no decisive swing in the median line, and this factor cannot be advocated at the present time as a significant technique in selection of candidates.

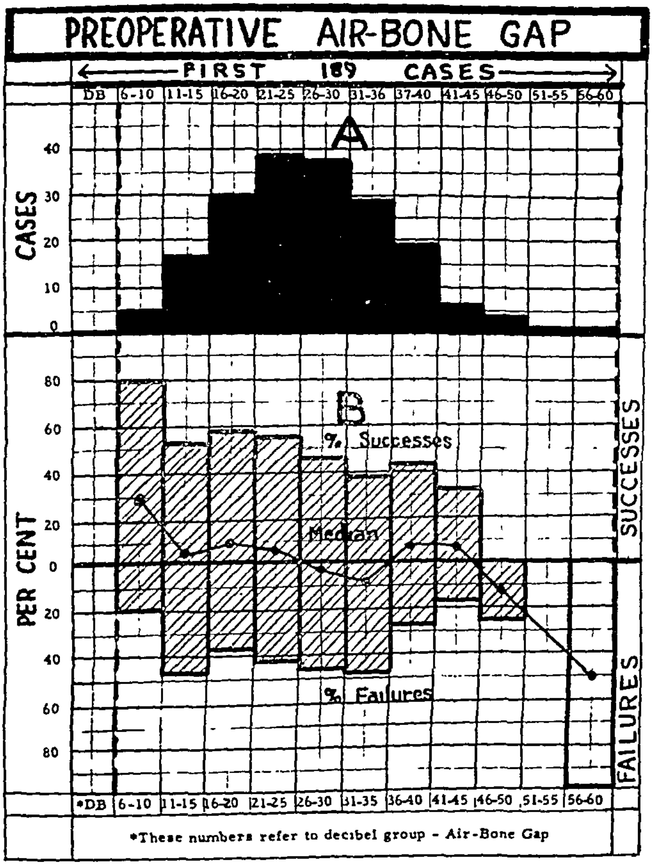


Fig. 21. Preoperative air-bone gap in 189 consecutive cases.

A—Shows the distribution of the differences between air and bone conduction before surgery.

B—Shows the per cent of successes in each 5 db group plotted above zero, and the failures in the same groups plotted below the zero line. Between minus 10 and minus 40 db the "median" line is nearly random in excursion.

The factor of age of candidate appeared to be a reasonable one in the question of selection for stapedolysis, and this factor is studied in Fig. 22. Fig. 22-A shows the percentage relation of success and failure as in previous graphs. There seems to be a trend showing a probability of greater success in the early 30 group and in the late 50 group, with a somewhat declining median line in the 40 to 50 age group; however, these trends are only of interest, and not significant enough to warrant our using them in candidate selection at the present time.

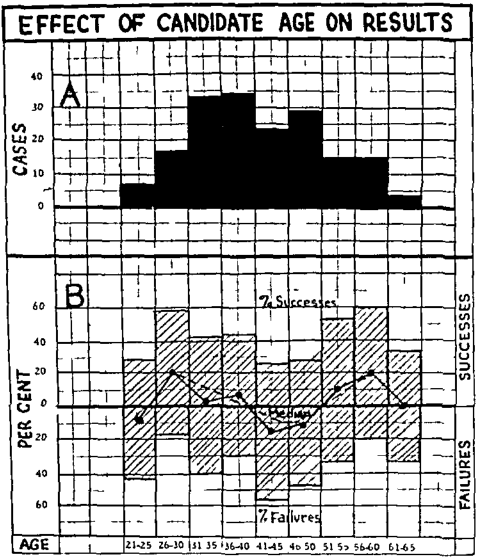


Fig 22 Effect of candidate age on results

A—Distribution of age in 176 candidates Information on age was lacking in 13 of the 189 cases

B—The median line in this case appears to display a definite characteristic The 26 to 30 and 56 to 60 year groups appear most favored The 40 to 50 year groups show least probability of success

VII. SUMMARY.

1. The primary direct surgical approach to the oval window has been revived in the treatment of otosclerosis.

2. Early reports of results in this renaissance of stapes mobilization (stapedolysis) surgery are encouraging.

3. Stapedolysis, a direct approach with no physiological deficit, has a wider application than fenestration in otosclerotic patients.

4. Successful results in stapedolysis are defined in two ways: *a.* achievement of 30 db level and *b.* eradication of bone-air gap.

5. Stapedolysis is an approach requiring varying techniques to cope with pathologic variations.

6. Precise microsurgical technique controlled by surgical audiometry is necessary in stapedolysis.

7. Operative and postoperative problems and complications have been analyzed in over 200 cases.

8. A significant number of successful results have been observed for periods of six to 12 months, as stable gains. Further long range observation is essential.

9. This technically complex and delicate surgical approach is, nevertheless, benign for the patient; it is quite free of operative and postoperative dangers.

10. In the evaluation of the most recent group of 89 consecutive cases (of a total of 189 cases), results show 56 per cent successes and 11 per cent partial gains.

11. These encouraging results seem to indicate that stapedolysis is a justifiable first choice in the surgical treatment of otosclerosis. In the event of failure, recourse to a successful fenestration is still possible.

12. This revival of stapedolysis will call for vigilant objectivity in long range appraisal.

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A STUDY OF THE ROLE OF CERTAIN FACTORS IN THE DEVELOPMENT OF SPEECH AFTER LARYNGECTOMY: 1. TYPE OF OPERATION; 2. SITE OF PSEUDOGLOTTIS; 3. COORDINATION OF SPEECH WITH RESPIRATION.†

Part 2: Site of Pseudoglottis.†

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Interest in discovering the site or source of vibration in substitute voice production has led to speculation and investigation from the time that cases of substitute voice were first reported. No definite site has been generally agreed upon, however, partly because of the unique difficulties encountered in efforts to study a pseudoglottis in action.

The value of objective evidence in demonstrating the location of the pseudoglottis is apparent. Not only is such evi-

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† This paper, presented in three parts, is based on the research associated with " A Study of the Role of Three Factors in the Development of Speech after Laryngectomy: Type of Operation, Site of Pseudoglottis, and Coordination of Speech with Respiration," an unpublished Ph.D. dissertation (Northwestern University, 1954) by Evelyn Y. Robe. Part 1 appeared in the March, 1956, issue of The Laryngoscope, and the May, 1956, issue will carry Part 3.

The research for the study was undertaken at the suggestion of Dr. Chevalier L. Jackson, Philadelphia, and was supported by a grant from the Illinois Division of the American Cancer Society.

Credit should be given to Mr. William W. Waldrop, Director, Speech and Hearing Rehabilitation Service, St. Luke's Hospital, for his advice and assistance in this study.

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dence important to surgeons who hold the opinion that every effort should be made to preserve the structures involved, but it would also unquestionably influence terminology and teaching procedures.

Gross classifications of substitute voice, such as the following, imply that the pseudoglottis is to be found in the general anatomical area named; however, a more accurate anatomical localization in some types of substitute voice seems desirable.

The Buccal Voice. This form must be distinguished from the "buccal whisper" produced without a pseudoglottis, which is almost completely inaudible and, therefore, considered generally undesirable. The true buccal voice is audible, its sound being produced by a pseudoglottis within the buccal cavity. In one type, an air bubble is formed between the cheek and maxillary arch. Muscular action drives the air through a narrow opening between or behind the teeth into the wider cavity of the mouth where the sound is articulated into speech.[23]

The Gastric Voice. According to Kallen, "in gastric voice the gastric air bubble functions to produce vicarious speech", frequently giving the auditory impression of "belly clang". The sound made by eructation from the stomach, combined with articulation, may be enough to eliminate the need for a pseudoglottis as a specifically developed structure in many laryngectomized speakers.

The Pharyngeal Voice. In this voice, the sound producing mechanism lies in the mesopharynx or hypopharynx.[10]

The Esophageal Voice. Sound for this voice is produced by a pseudoglottis formed at the upper end of the esophagus at the level of the cricopharyngeus muscle. The air reservoir is within the lumen of the esophagus and the cardiac end of the stomach.[10]

The possibility that transition from one type of pseudovoice to another may occur has been discussed by Kallen. When first learning to speak, the beginner overdoes the process of taking in air, a large supply accumulating in the stomach and lower esophagus. As he continues to practice, however, the air is converted into sound more quickly, the path becoming ever shorter, until the air is transformed automatically into sound with the swallowing.[16]

While "esophageal voice" is the term most frequently used to describe substitute voice, usually without particular reference to the site of pseudoglottis, some investigators prefer a less specific term, such as "substitute voice". Kallen recommends the following:

> "Since the mechanism of speech varies so greatly in laryngectomized subjects, a term is preferred which designates all the facts. Although *pseudovoice* does so for voice, it is inapplicable to the pseudowhisper. I propose, therefore, the term alaryngeal speech, which means speech without a larynx, regardless of the mechanism involved, and which logically includes the pseudowhisper."[10]

As mentioned earlier, efforts to study the site of the pseudoglottis invariably meet with great difficulty and lead to results which are generally inconclusive. When mirror examination of the lower pharynx was attempted, for example, movements of gagging and the presence of secretions often obscured the vibrating structures. Protrusion of the tongue for viewing the hypopharynx ordinarily modifies or eliminates posterior glossal vibration, although in some cases inspection of the area during sound production is possible without tongue protrusion. Froeschels cautions that even when the back of the tongue is retracted toward the pharyngeal wall or the narrow pillars of fauces appear to be vibrating, there is still the possibility that the real pseudoglottis is located at a lower level. He adds that "even if stroboscopy reveals vibrations of the pillars or of the tensed velum palatini, it still cannot be decided whether or not these vibrations are transferred by air originally brought into vibration at the entrance of the esophagus."[3] With regard to other methods of study, Kallen suggests:

> "Palpation also is an aid in differentiating areas of vibration. And although the roentgen rays are not useful here as in the study of the vicarious air chamber, they occasionally enable one to make certain inferences. In general, however, the membranous folds and the muscular bulgings involved are too slight to be distinguishable from surrounding tissue. The findings in a few patients who had come to necropsy because of

recurrence have not helped much in indicating how the vicarious glottis developed and acted."[10]

Various anatomic structures which may serve as a pseudoglottis have been reported in the literature. Stern thought it could be formed in any one of the following places: *1.* between the base of the tongue and the posterior wall of the pharynx; *2.* between the back of the tongue and the tightly stretched velum; *3.* between strongly contracted posterior arches of the two sides; *4.* between contracted portions of the inferior constrictor; *5.* between the epiglottis and the two folds formed laterally from the musculature of the pharynx; *6.* the esophageal mouth of Killian, between the folds of the cricopharyngeus; *7.* no real glottis, in some, rather the sound of eructated air.[17] Kallen lists the same structures, except the last. He adds that "every fold of mucous membrane, every favorably placed cicatricial band, every muscle or muscular remnant, may serve as the basis for the development of a pseudoglottis."[10]

Jackson and Jackson report that the tissues they have observed in vibration include:

"*1.* The pillars of the fauces under muscular action; *2.* the tongue against the back wall of the pharynx; *3.* the tongue against the posterior pharyngeal wall; *4.* the remnant of the tip of the epiglottis against the back wall of the pharynx; *5.* adventitious folds in the hypopharynx; *6.* the anterior wall of the hypopharynx coming against the posterior wall; *7.* a pseudosyrinx formed in the esophagus by the collapse of one wall against the other with the axis of the chink noted in some cases in the frontal plane, in others in the sagittal plane, and in still others in one of the diagonal planes; *8.* a cicatricial band against the normal tissue; *9.* a pair of cicatricial bands; *10.* secretions forming bubbles aiding the tissues."[9]

Levin thinks that the pseudoglottis varies in the individual patient, depending upon the type of operation performed and the remaining tissues. The structures which may function as vibrators are:

"*1.* the epiglottis tip, if this is permitted to remain; *2.* the pillars of fauces; *3.* the tongue against the posterior wall;

4. the collapsing esophageal wall against the opposite side; *5.* a cicatricial band against the normal tissue; *6.* others which cannot be identified by present knowledge."[11]

Mason also describes these structures as possible sites of the pseudoglottis, but adds that "more often, no definite pseudoglottis can be demonstrated even though the voice may be perfectly satisfactory."[14]

A considerable number of investigators are of the opinion that the mouth of the esophagus, or cricopharyngeus, forms the most efficient pseudoglottis for the laryngectomized speaker. Included in this group are: Kallen,[10] Stetson,[22] Gatewood,[6] Howie,[8] McCall and Stover,[15] Greene,[7] Martin,[13] Levin,[11] Lindsay, Morgan and Wepman,[12] Mason,[14] Moolenaar-Bijl,[16] Pancoast, Pendergrass and Schaeffer,[20] Gardner,[4] Negus,[19] and Brighton and Boone.[2]

Kallen reported that as early as 1888, Bandler described a case in which the cricopharyngeus functioned as the pseudoglottis in producing a substitute voice. In the same article reference was made also to Fraenkel in 1893, who published an account of a similar case which had been corroborated by Gutzmann.[10]

Seeman made one of the first scientific studies of the problem, using the method of Roentgen examination. Morrison reported Seeman's conclusions to the effect that the anatomic and physiologic conditions of the esophagus meet the requirements for the production of substitute voice in every respect. He described contractions of the esophageal mouth during phonation and expressed the opinion that the striated muscles involved could function under partial voluntary control. The esophagus formed an ideal air chamber, he thought, because of its capacity to: *1.* hold more than a few cubic centimeters of air; *2.* be filled with air quickly and voluntarily; *3.* expel air under control; and *4.* set the pseudoglottis into vibration by providing the necessary air current thus fulfilling the requirement that the air chamber be caudal to the pseudoglottis.[17]

Morrison and Fineman described the pseudoglottis formed at the cricopharyngeus and its advantages in the following way:

"True esophageal voice is characterized by the fact that the pseudoglottis lies at the level of the cricopharyngeus muscle, and the air reservoir is within the lumen of the esophagus and the cardiac end of the stomach. . . When it has been learned and practiced long enough it is produced with little effort, is loud and clear enough for all ordinary conversation and carries very well by telephone."[18]

The majority of those who regard the cricopharyngeus as the most frequent site of the pseudoglottis base their conclusions, at least in part, on Roentgenological observations, which despite many limitations, have provided the most objective visual evidence thus far.

The studies of Brighton and Boone in 1937, Pancoast, Pendergrass and Schaeffer in 1940 and Lindsay, Morgan and Wepman in 1944, were specifically devoted to Roentgenologic and fluoroscopic examination of the substitute speech mechanism in laryngectomized persons.

Brighton and Boone reported that their films showed the site of vibration or pseudoglottis as corresponding to the anatomical position of the cricopharyngeus muscle; moreover, *they stated that "the action of the pseudoglottis is similar to* that of a normal glottis, the vibrating membrane of the tracheobronchial tree of certain lower animals or the reed of an artificial larynx".[2]

Pancoast, Pendergrass and Schaeffer observed that:

"As the patient begins to talk, certain muscles appear to contract and reduce the shadow of the pharyngeal air space. In this stage it is possible to see a soft-tissue shadow, which more or less completely separates the air-space of the pharyngo-esophageal reservoir from that of the remainder of the esophagus. We have felt that this shadow is produced by a compensatory development of the cricopharyngeus muscle."

Lindsay, Morgan and Wepman discovered in the course of their investigation that:

"During phonation it can be seen that the sphincter is not completely closed, leaving a small channel for air to escape. Although the pitch of the sound produced possible to detect vibratory movement of the muscular fold under the fluoroscope; however, with an exposure which varies from one-twentieth to one-thirtieth of a second, the Roentgenograms taken during phonation regularly show distinct blurring of the edges of the fold, while those made during resting stage or modified Valsalva test show clear-cut borders and a completely closed sphincter. It appears clear, therefore, that the movement set up in the borders of the muscular fold by the column of air escaping through the spincter constitutes the mechanism of sound production."[12]

They concluded from their data that "the proficiency of the esophageal voice is related to the degree of control over the cricopharyngeus muscle".[12]

One of the most recent investigations of the subject based almost entirely on fluoroscopic and Roentgenographic data is that of Pellegrini and Ragaglini, in 1951. Their observations were made on laryngectomized patients, before and after speech training. In contrast to the majority of investigators, they refute the idea that a pseudoglottis actually forms in the speaker. They conclude that a squeezing mechanism apparently forces air to pass through a narrow channel, thus producing sound.[21] No explanation is given as to why the narrow channel should not be considered the pseudoglottis.

While fluoroscopic and Roentgenological studies have provided the most objective data thus far, the need for further research in this area, employing new or different techniques is evident. In the study being reported here motion picture photography and Roentgenography were the procedures used to obtain visual evidence of the location of the pseudoglottis and its air supply.

Twenty laryngectomized speakers who exemplified variations in the manner of producing voice (*e. g.*, lip and swallowing movements), as well as differences in pitch, were selected as subjects. A thorough examination of the pharynx and hypopharynx of each subject was made with a conventional laryngeal mirror in order to observe size, shape and functioning and to note the presence or absence of structures such as tonsils and epiglottis. Each operative report had previously been reviewed to learn what muscles or structures remained which might function in the production of sound. The subject was then asked to produce the vowel "eh": first, in his usual manner; and again with the tongue forward and the mirror adjusted in the pharyngeal area to reflect vibratory movement there or below.

When the examination indicated that the individual would be a satisfactory subject, or that with training he might become one, he was given equipment and instructions for practice at home. The equipment consisted of a No. 3 laryngeal mirror and two stationary mirrors (one a concave head mirror), mounted on a ring stand. Directions were given for the location of the light to be reflected from the concave mirror into the pharynx, the adjustment of the guttural mirror, and for checking the procedure in the second stationary mirror. After adequate practice at home and in the laboratory, the subject was considered ready for photography.

The procedure followed was the same as for laryngeal photography.[1] The subject was instructed to produce the vowel "eh", sustaining it as long as possible or continuing to repeat it while the camera was in operation. Magnetic tape recordings of the sounds produced were made concurrently with the films.

Analysis of the films consisted essentially of location of the sound source and descriptions of the individual pharyngeal variations.

Original plans included the use of high-speed photography, but technical difficulties made it impossible to carry out this procedure. The laryngectomized patients were not able to devote the time necessary to learn to produce voice upon a given signal, with the pharyngeal mirror in position and within the brief time of the filming.

X-ray Data: Sixteen laryngectomized speakers served as subjects for the Roentgenography. For visualization, a mixture of barium and butter was made, as described by Hoerr[5] and was spread thickly on a large soda cracker. This combination adhered to the mucosal surfaces a sufficient length of time to permit good Roentgenograms. Half the cracker was consumed before the first two films; the remainder before the others.

Film 1 was made with the subject at rest, prior to air intake for speech; Film 2 showed the structures just after the subject had taken in a maximum amount of air in preparation for phonation. Film 3 was exposed during production of the vowel "ah" and Film 4 followed immediately after the production of the vowel.

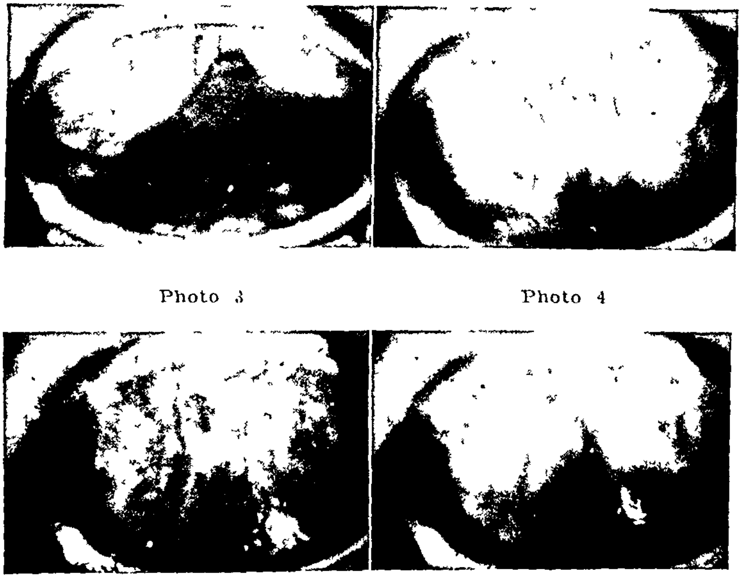

Photo 1

Photo 2

Photo 3

Photo 4

Fig 1. Photographs 1, 2, 3, 4 Enlargements of representative 16 mm. motion picture frames showing sphincter-like constriction and relaxation of the pharyngeal walls during bucco-esophageal phonation

The subject's position was changed for the fifth film so that he stood at a 45-degree angle to the chest table. This provided a full length view of the esophagus. The instructions given were the same as for Film 2 and the exposure was made when the subject had taken in the amount of air preparatory to phonation.

Areas of constrictor action, changes in contours, or other evidence of pseudoglottis formation were studied, and locations in relations to vertebrae were noted. Visible changes in the air column in the pharynx and esophagus preceding phonation, and presence or absence of an air column below the pseudoglottis when phonation was initiated and immediately afterwards were also noted.

Films 14 x 17 inches, which showed an increased length of esophagus, were studied separately to discover whether the change in the subject's position might bring into prominence structures or evidence of function which were not visible in the lateral view.

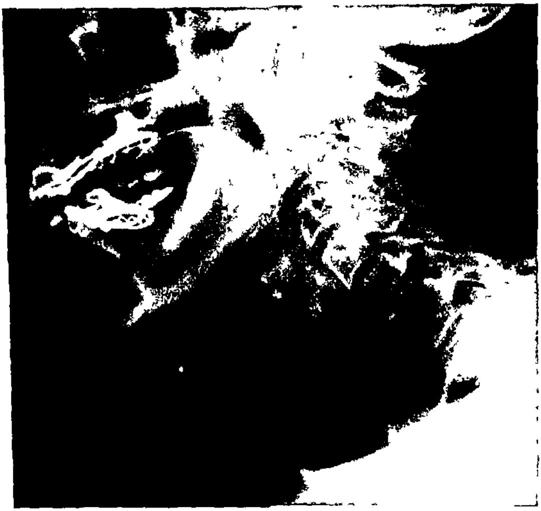

Fig 2 Subject XI, Film No 1 Air column observed in esophagus preceding phonation

A study of the motion picture films, the associated sound recordings and the X-ray photographs pointed up certain problems and provided a few conclusions.

Motion Picture Data. Only eight of the 20 subjects originally selected for photography finally proved to be suitable. The major difficulties were: some could not phonate with the mouth wide open, others could not tolerate a mirror, while still others were unable to produce sound with a mirror in place.

In the analysis of the films, the pattern of movement most frequently observed was that of a circular drawing together of the walls of the pharynx, and a marked bubbling of saliva as the sound was produced, followed by a drawing apart of the pharyngeal walls. These adjustments are illustrated in Fig. 1 by selected frames from a film sequence. In Photograph 1, the irregular mass occupying the upper third of the image is the base of the tongue. The dark area just above the lower part of the mirror rim represents the posterior pharyngeal wall which drops almost vertically. Photographs 2 and 3 show a rapid backward sweep of the tongue combined with the

Fig 3 Subject XI Film No 2 Air column following maximum oral intake of air preparatory to sound production

radial movements of the lateral pharyngeal walls in a sphincter-like closure. The bright spot in the lower section of Photograph 3 pictures a bubbling of mucous and saliva produced by the expulsion of air from below it. It is probable that some of the sound is produced by this bubbling action. Photograph 4 shows the tongue and pharyngeal walls returning to the position of rest shown in the first photograph.

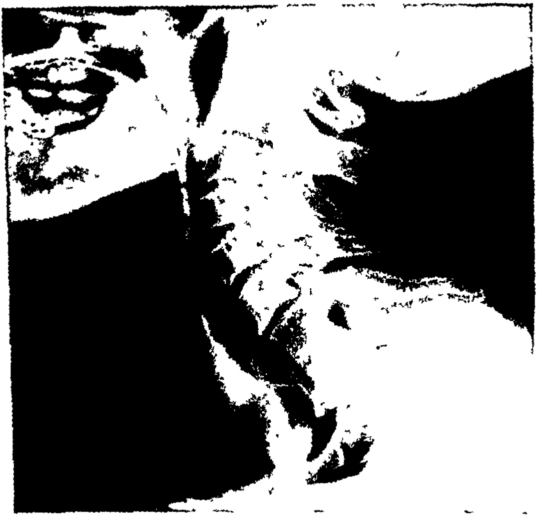

Fig 4 Subject M, Film No 3 Constriction of the esophageal air column during production of vowel sound 'ah'

The photographs of Fig. 1 show a type of closure which involves the tongue and which is made relatively high in the pharynx. The closure in most of the subjects was lower in the pharynx, but these photographs were used because they gave a clearer representation of the movements.

The eight individuals participating in this procedure were excellent speakers. Their films indicate that in every case, with the possible exception of one, the pattern of movement as described above takes place at the level of the cricopharyngeus. This evidence, although from a limited number of subjects, lends further support to the observations of those using other methods to determine the site of the pseudoglottis, that the region of the cricopharyngeus is the usual location.

Fig. 5. Subject XI, Film No 4. Constriction still apparent following phonation, air column below decreased in size.

Perhaps the chief value of the results of the pharyngeal photography is that the films demonstrate that clear, permanent, visual records of the changes taking place in the mouth, pharynx, and upper esophagus during production of substitute voice are possible.

X-ray Data: An air column was observed in the pharynx and esophagus of 15 of the 16 subjects preceding phonation (see Fig. 2, Subject II, Film 1). An increase in the size of the air column was noted in 11 instances, following maximum oral intake of air preparatory to sound production (see Fig. 3, Subject II, Film 2). During the production of the sustained vowel "ah" in Film 3, definite constrictions of the esophageal air column were visible in the films of 13 of the subjects. In seven of the 13, the constrictions appeared in the area of the esophagus opposite vertebrae 5 and 6. In three, it was opposite vertebra 7. In one case, it was opposite the third vertebra, and in two cases, a double constriction was noted.

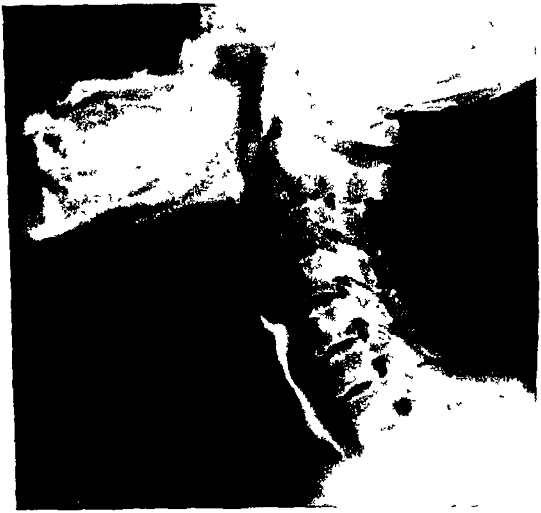

Fig 6 Subject XV, Film No 1 Beginning of phonation of poor speaker. Note absence of air column in cervical esophagus

The appearance of such constrictions, particularly in the region of the fifth and sixth vertebrae, bears out the findings of other investigators who have used the Roentgenological method. The present X-ray films also tend to confirm Hoerr's[5] observation that the laryngectomized speakers with the best voices have the sharpest constrictions in the area of the cricopharyngeus.

In most subjects the constriction was still apparent following phonation, but the air column below had decreased in size (see Fig. 5, Subject XI, Film 4). In some cases, the column above the constriction had grown larger.

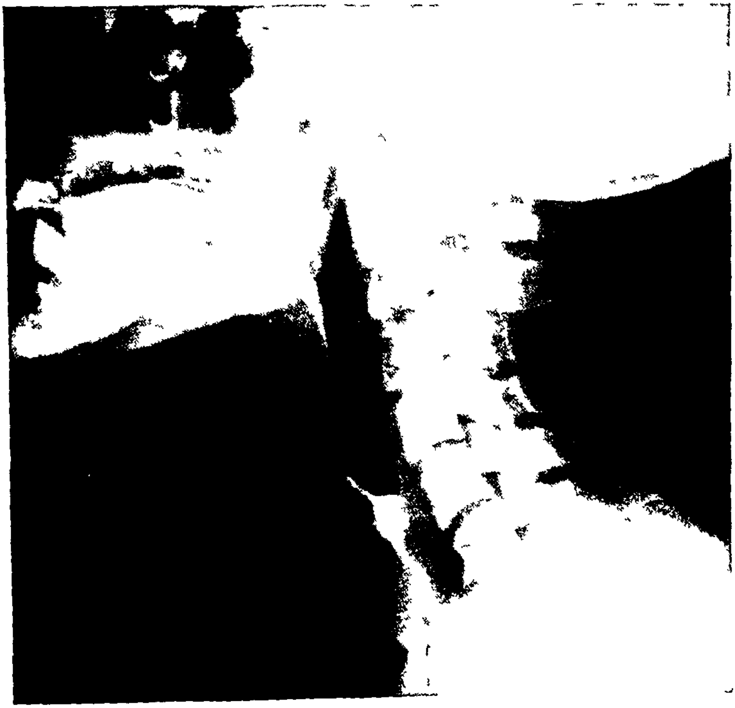

Fig 7 Subject XV, Film No 2 Second stage of phonation of poor speaker Tongue is raised, pharynx narrowed

One of the most significant observations provided by the X-ray series relates to the presence or absence of an air column below the constriction as described for Subject XI, Film 3 (see Fig 4). In the 14 good speakers, a sizable air column was demonstrated below the area of constriction or pseudoglottis. In the two poor speakers, one whose X-rays are shown, this air column was not demonstrated, Subject XV (see Figs. 6, 7, 8, 9). It appears that an adequate sized air column is extremely important.

The lateral view X-ray films of the two subjects demonstrate the difference which is visible when an air column is present and when it is not. Subject XI is a speaker with an excellent substitute voice of more than adequate volume, while Subject XV is a poor speaker who lacks control of the air used for sound, and whose voice is frequently too weak to be heard. It should be noted that in Subject XI the hyoid bone has been

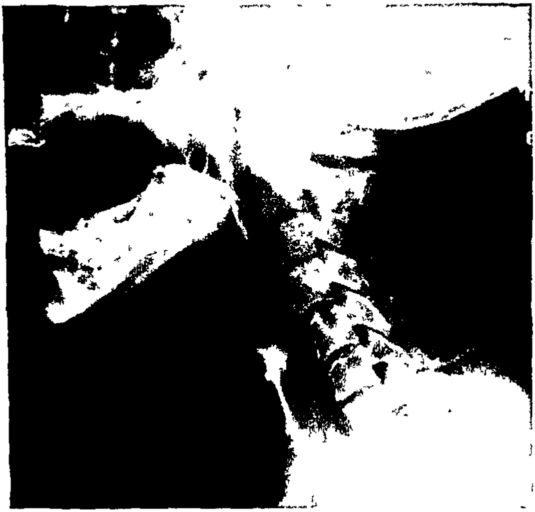

Fig 8 Subject XV, Film No 3 Mouth open, pharynx constricted, esophageal column compressed from below

left in place; in subject XV the hyoid bone has been removed. Of the remaining speakers, those possessing superior voices generally revealed a larger air column below the constriction during phonation (see Fig. 4) than that which appeared above. The presence or absence of the hyoid bone was not related to quality of voice in the other subjects.

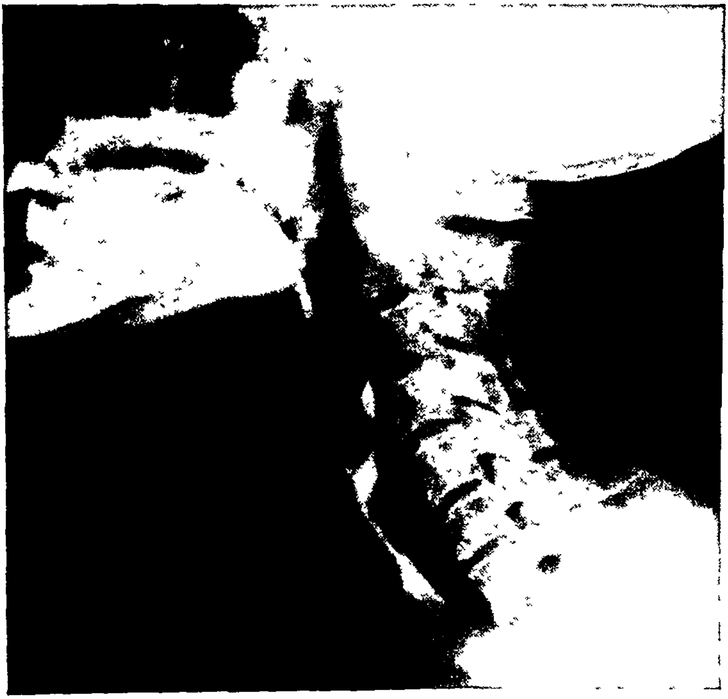

Fig. 9. Subject XV, Film No. 4. Completion of phonation of "ah." Mouth closed, pharynx returning to resting position, esophageal column returning to original straight course

The single 14x17 inch oblique chest view X-ray films yielded little information beyond the observation that with the exception of two subjects, who were very poor speakers, air was visible throughout the length of the esophagus rather than in the upper part alone.

In summary, it appears that two factors are present in the good speakers: *1.* A well defined controllable constriction zone in the region of the cricopharyngeus which will act as a pseudoglottis; *2.* an adequate sized air column below the pseudoglottis.

SUMMARY.

The location of the pseudoglottis in the laryngectomized speakers was objectively visualized by pharyngeal motion picture photography and Roentgenography.

The pharyngeal films which were made during the production of sound revealed a pattern of movement characterized by a circular sphincter-like drawing together of the walls of the pharynx, a bubbling of saliva as the sound was produced, followed by a drawing apart of the pharyngeal walls. This pattern of movement appeared to originate at the level of the cricopharyngeus.

Analysis of a series of four 10x12 inch lateral view X-ray films taken before, during and following phonation resulted in the following observations:

1. A constriction of the pharyngo-esophageal air column was present in all good speakers.

2. The constriction appeared most frequently at the approximate level of the fifth vertebra and the cricopharyngeus muscle.

3. An air column of adequate size below the area of constriction or pseudoglottis was observed in the good speakers and not in the poor speakers.

4. The formation of this column of air below the pseudoglottis, when sound is produced, appears to be analogous to the utilization of subglottal air in normal phonation.

The 14x17 inch oblique X-ray films, made at the point of maximum intake of air for speech, revealed that air is generally visible throughout the length of the esophagus, rather than in the upper part alone.

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School of Speech, Northwestern University, Evanston, Ill

AUDIOLOGY FOR INDUSTRY

Colby College, Waterville, Maine, presents the Fourth Annual Course in Industrial Deafness, August 5 11, inclusive Objective of the course will be to train personnel in initiating and in conducting hearing conservation programs in noisy industries Seven full time instructors have been selected from authorities in this field Class limited to approximately twenty participants

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PURE TONE THRESHOLD AND HEARING FOR SPEECH—DIAGNOSTIC SIGNIFICANCE OF INCONSISTENCIES.*

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Louisville, Ky.

The lack of correlation between pure tone threshold and speech hearing may be directed in either of two ways. Hearing for speech may be worse than would be anticipated from the pure tone threshold audiogram, or it may be considerably better. Most inconsistencies observed are in the direction of speech hearing as not being so good as the pure tone threshold would indicate. This will be discussed briefly, and then the reverse relationship will be considered at greater length.

SPEECH HEARING WORSE THAN PURE TONE THRESHOLD.

In pure conduction deafness, the hearing loss for speech correlates very well with the pure tone threshold. In neural deafness, the correlation may be fairly good in some instances, but frequently the difficulty in hearing and understanding speech is much greater than is indicated by the pure tone curve.

An extreme example of the latter type is cited to illustrate the absolute necessity of doing speech tests in evaluating the hearing capacity in cases of nerve deafness (see Fig. 1). This patient was referred to me by a hearing-aid dealer who could not understand why the patient was not able to use a hearing aid in the right ear, and why only mediocre benefit was obtained from using the aid in the left air. Speech tests revealed that the patient could understand only occasional spondees in the right ear and the discrimination score in this ear was 0 per cent.

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There was marked recruitment in this ear and low frequency tinnitus was present. This was unquestionably an end-organ lesion, most likely of the hydrops type. Extreme loss of speech hearing in the presence of only moderate, flat pure tone loss is fairly common in cochlear or labyrinthine hydrops. The spondee threshold in the left ear was 50 db and the best discrimination score was 68 per cent. Recruitment in this ear was moderate in degree.

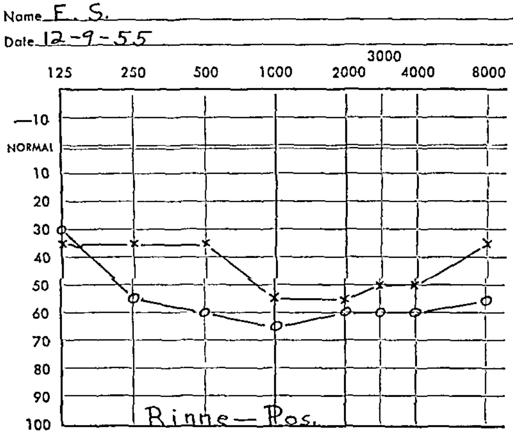

Fig 1

Much remains to be learned concerning the nature of physiological disturbances of end-organ function which are related to speech-pure tone discrepancies. Harris et al.[1] did a detailed study in a small group of cases, and noted that recruitment seemed to be a most important factor in such discrepancies in younger individuals. Pitch deterioration alone was not important; in fact, if recruitment was accompanied by pitch deterioration, speech-pure tone correlation was usually fairly good. The reason for this last relationship is not known.

It is not difficult to understand the importance of recruitment in speech-pure tone discrepancies if the factor of "narrowed dynamic range" expressed by Eby and Williams[2] is considered. They noted that speech was intelligible over a rather narrow range of intensity above threshold in an ear with recruitment, in contrast to a non-recruiting ear. The phonetic elements of words and sentences must vary considerably in intensity, even under calibrated laboratory conditions. The elements with greater intensity may then fall within the recruited range and seem unusually loud, while the less intense elements may be barely or not quite audible. Consequently, in an ear with considerable recruitment this abnormally wide range of loudness must create a state of utter confusion; *i. e.*, some elements of the presented speech seem too loud and others are scarcely audible. If for example the loudness range of ordinary speech elements just above threshold perceived in a non-recruiting ear is only 20 db, the perceived loudness of similar speech elements at 20 db intensity above threshold in an ear with recruitment has a much wider range (see Fig. 2).

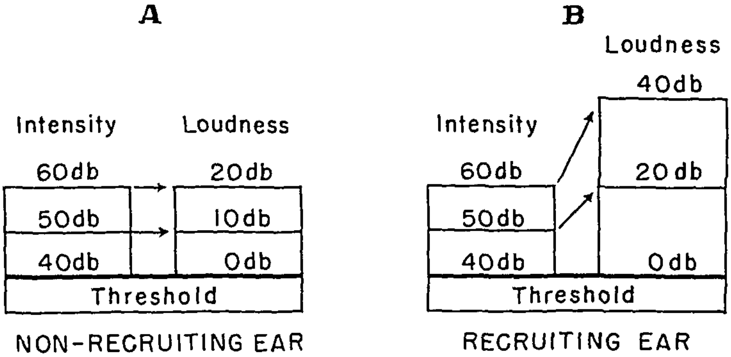

Fig. 2. These diagrams represent the relationship of loudness to intensity in a non-recruiting (A) and a recruiting ear (B). A threshold of 40 db. is indicated for each ear. In the non-recruiting ear the increase in loudness above threshold is always directly proportional to the intensity increase; i. e., when the intensity applied to the ear is 50 db. (10 db. above threshold) the loudness experienced is 10 db. At 60 db. intensity (20 db. above threshold) the loudness is 20 db. In B is represented an ear with moderate recruitment and also a threshold of 40 db. When a sound intensity of 50 db. (10 db. above threshold) is applied to this ear, the loudness experienced is 20 db. Each increase in intensity in this ear shows a disproportionately greater increase in loudness. The rapidity of increase of loudness depends on the degree of recruitment present and varies considerably from one case to another.

As a result the intensity range which permits intelligibility of speech in an ear with recruitment is "narrowed".

Poor speech hearing is also to be expected when there is a rather abrupt drop in the audiometric curve for the higher speech frequencies, resulting in consonant speech elements not being heard. In such cases hearing for spondees may correlate fairly well, but discrimination is usually impaired. In older persons, the capacity to understand speech is usually much poorer than would be suggested from the audiometric curve. The lesion here is probably central as well as peripheral. A recent study by Bordley and Haskins[3] reveals that in such instances the PGSR shows better hearing for pure tones than does standard subjective audiometry. They considered this to be evidence of cerebral sclerosis. Experimental evidence indicates that cortical integrity is not essential for a good PGSR response. Such individuals were unable to derive any benefit from a hearing aid. Gaeth[4] has referred to this impairment of auditory function in older individuals as phonemic regression. Actually, this is probably more of a perceptive deterioration than a regression. Recruitment is not always present in presbycusis.[5] It is possible that the degree of recruitment found might be an indication of the end-organ component in the total perception problem in presbycusis.

Tinnitus frequently interferes to some extent with good speech hearing. This is particularly true of a low-pitched tinnitus, such as is frequently seen in cochlear hydrops.

SPEECH HEARING BETTER THAN PURE TONE THRESHOLD.

This relationship is relatively uncommon in private practice. Audiological observations during and after World War II indicated that such cases are on a psychogenic basis. The occasional cases that I have seen in private practice are apparently in the same etiological category. As a rule these patients have a moderately severe pure tone loss on subjective audiometry, and yet can carry on conversation at a distance of several feet with very little difficulty; this is the clue to the diagnosis. Calibrated speech tests will then verify the discrepancy between pure tone threshold and hearing for speech.

I have in the past few years observed several school children in whom the pure tone loss was much greater than the loss for speech. This relationship in adults in private practice is very infrequent. The details of one case will be cited to illustrate the clinical aspects of this problem.

A 12-year-old girl was referred to me because she was not doing well in school. An audiogram made by the school nurse showed a 55-60 db. loss in both ears. My initial audiogram on Feb. 4, 1955, substantiated this finding (see Fig. 3). Yet this girl could repeat words heard at a distance of 25 feet. Her otolaryngology examination was negative. A diagnosis of psychogenic deafness was made, and thorough psychological study was recommended. A follow-up hearing study was done on May 4, 1955 (see Fig. 2). There was still a moderate loss on pure tone examination. Hearing for speech showed normal threshold for spondees and a 100 per cent discrimination score. Because of uncertain calibration of the masker, a D-S test was not done. A recheck of an audiogram on Dec. 3, 1955, showed an almost normal pure tone threshold. At this time the patient seemed to have a more normal psychological attitude in contrast to a somewhat withdrawn type of personality noted at the time of her initial examination.

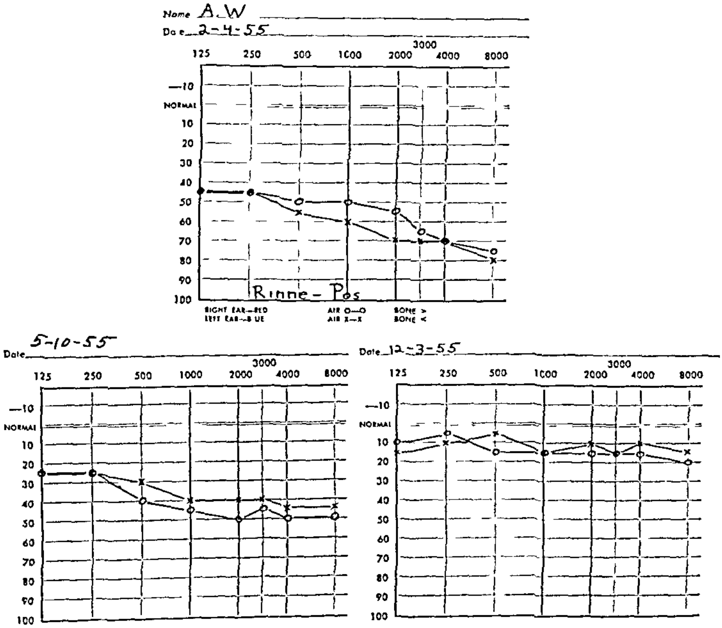

Fig. 3.

A brief review of her psychological background revealed that the girl had been rejected by her parents at the age of one year and placed in a foster home. At the age of three she came to live with an aunt with whom she lives at the present time. The aunt states that at the age of three the girl was not inclined to play much with other children and frequently sat by herself, talking and making facial grimaces. As far as is known she developed speech at the usual age. Shortly before the time I first saw her she had had a severe conflict with her teacher. This may have been the precipitating factor for her psychogenic deafness. When last seen recently, she was again well-adjusted in school, and it will be noted the audiogram is near normal.

In many cases the psychological problem is not as apparent as it was in this instance. It is doubtful whether a detailed psychological study is necessary or advisable in all cases; however, a superficial survey should be made, and if there is evidence of any serious difficulty, adequate psychiatric evaluation should be recommended. The otologist must use his clinical judgment on this aspect of the problem after he has completed his study.

To my knowledge there has to date been no attempt made to account for the disproportionately greater loss for pure tones than for speech in cases of psychogenic deafness. An attempt will be made here to supply a hypothesis to explain the genesis and basis of this clinical entity.

Myklebust[6] has shown that some children with otherwise normal hearing show very poor conscious response to pure tone stimulation. He explains this on the basis that a pure tone is too abstract and meaningless to arouse cortical evidence of recognition on the part of the child. He states that in the process of auditory maturation the child first shows evidence of response to speech or complex meaningful sound or noise, and that visible evidence of response to pure tones is, in some instances, delayed. Some children respond to pure tones at a much earlier age than others. This does not mean that the organ of Corti or the central pathways are not stimulated by a pure tone but merely that recognizable conscious evidence of response is delayed in developing.

It is an accepted fact that some predisposed individuals will regress to infantile patterns psychologically in certain areas of their behavior when subjected to undue stress.[7] Since the function of hearing represents to some extent a psychological reaction, it is possible that some individuals are so sensitized or conditioned as to regress in the auditory area under certain situations of psychological stress. In other words, if an individual regresses with respect to his auditory function, he would lose first that facet of function which was last to develop in maturation; *i. e.*, conscious recognition of pure tone stimulation. If regression is severe, a moderate or severe loss for speech and other sounds as well as for pure tones will occur.

My first thought was to call the *modus operandi* of this concept of psychogenic deafness a "dematuration"; however, I think that a more appropriate and descriptive term would be "psycho-auditory regression". I believe that the greater loss for pure tones in these cases would more logically be a regression than a suppression.

DISCUSSION.

Pure tone speech hearing discrepancies are not present in pure conduction deafness. When the pure tone speech discrepancy is marked, it is usually obvious to the alert clinician on comparison of the pure tone audiogram with the patient's ability to understand conversational voice. Calibrated speech tests are necessary to establish a more exact determination of speech hearing capacity. The most common discrepancy is for speech hearing to be poorer than the pure tone threshold. Cochlear hydrops and some instances of presbycusis are particularly inclined to have considerable deterioration of speech hearing. A less common relationship is for speech hearing to be much better than the pure tone threshold. This last situation should make the clinician suspicious of an underlying psychogenic basis. If facilities are available, a D-S test and a PGSR study should be done in these cases. The PGSR (objective audiometry) would be expected to show a better threshold than standard subjective audiometry. It is hypothesized that psychogenic deafness can be explained on the basis of a "psycho-auditory regression" resulting from psychological stress in a predisposed or preconditioned individual.

SUMMARY.

1. Some form of speech testing is essential as a supplement to the pure tone audiogram in evaluating an individual's capacity to hear and understand speech.

2. Greater loss for speech than for pure tones is rather common in nerve deafness.

3. Greater loss for pure tones than for speech is highly suggestive of psychogenic deafness.

4. The concept of "psycho-auditory regression" as a basis for psychogenic deafness is proposed.

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THE AMERICAN OTORHINOLOGIC SOCIETY FOR PLASTIC SURGERY, INC.

A scientific meeting of the American Otorhinologic Society for Plastic Surgery will take place at the Waldorf-Astoria Hotel in New York City on May 18, 1956. An operative clinic will also be held during the morning of May 19th at the Montifiore Hospital.

The program will consist of medico-dental presentations and plastic surgery of the head and neck.

For further information write Louis Joel Feit, M.D., Secretary, 66 Park Avenue, New York 16, N. Y.

THE MECHANISM OF OPENING OF THE HUMAN LARYNX.*†

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MILOS BASEK, M.D.,
and
V. EPANCHIN, M.D. (By invitation),
New York, N. Y.

In a recent discussion concerning the mechanism of laryngea closure[1] certain concepts arising out of Roentgenographic investigations were presented. It was shown that a ball-valve type of mechanism occurred, produced by pressure of the pre-epiglottic body against the upper surface of the vestibular folds and brought into action when the distance between the larynx and the hyoid bone was reduced. Evidence was presented suggesting that extrinsic laryngeal muscles were responsible for this movement and were thereby able to close *the larynx even when the intrinsic muscles supplied by the* recurrent laryngeal nerves were paralyzed.

Electromyographic studies have produced additional evidence supporting these ideas. In conjunction with Roentgenographic and cinematographic analysis of vocal cord movements, the findings also indicate that current views on the mechanism of opening of the larynx and glottis[2] require modification. The evidence concerning the mechanism of opening is presented here.

METHODS.

The subjects were three male physicians in normal health.

Electromyography: Action potentials from laryngeal muscles were recorded during normal and deep respiration. The muscles studied were the sternothyroid, sternohyoid, thyro-

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hyoid, cricothyroid and vocalis, and the rectus and external oblique muscles of the abdomen. The muscles were identified by reference to their point of attachment to skeletal structures. Concentric bipolar needle electrodes were inserted through skin wheals made with one per cent procaine into the muscles in a plane closely parallel to the long axis of their fibers. Six muscles were studied simultaneously. The potentials were led to an eight channel Grass model III ink-writing oscillograph. The seventh channel recorded sounds through a microphone placed in the subject's mouth. The eighth channel registered a signal operated by the subject's hand and arranged to light simultaneously a lamp visible in the field of a 16 mm. motion picture camera. This camera photographed the movements of the anterior prominence of the thyroid cartilage. The signal was used to mark the start and end of each maneuver.

Roentgenograms: Lateral and anteroposterior exposures were made at the end of normal expiration, enforced expiration, and at the end of deep inspiration. The technique for lateral views was: tube film distance 72 inches, centered 1.5 cm. behind the thyroid cartilage notch, 72 kv., 400 ma., 1/10 second. The subject sat up with the head steadied by a strap. Anteroposterior laminograms were obtained in a plane 2 cm. behind the thyroid cartilage notch, tube film distance 30 inches, 50 milliamps for 3 seconds, 75 kv., patient recumbent.

Motion Pictures of Vocal Cord Movements: The larynx was visualized by a laryngeal mirror and photographed at 16 frames per second during phonatory and respiratory movements.

RESULTS.

Electromyograms: There is continuous activity of all the intrinsic (cricothyroid and vocalis) and extrinsic (sternothyroid, sternohyoid and thyrohyoid) muscles of the larynx studied, but the intensity of activity in the various muscles depends upon the laryngeal movement that is being performed.

In quiet respiration (see Fig. 1) there is some activity in all the muscles of the larynx throughout both inspiration and expiration.

With stronger respiratory efforts (see Fig. 2) there is increased activity in all the laryngeal muscles and a definite dif-

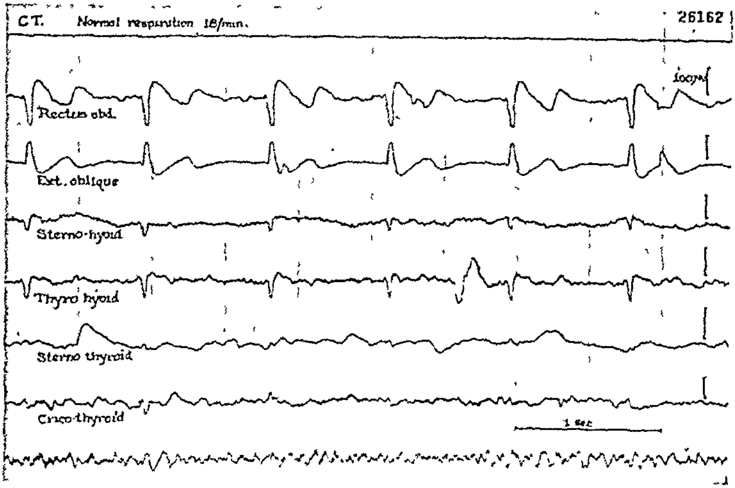

Fig. 1. Electromyograms of abdominal and laryngeal muscles during quiet respiration. Subject C. T. There is slight activity continuously in all the laryngeal muscles. The large deflections in the abdominal tracings are due to electrocardiogram pickup. The bottom tracing in Figs. 1 to 4 recorded currents from a microphone near the subject's mouth.

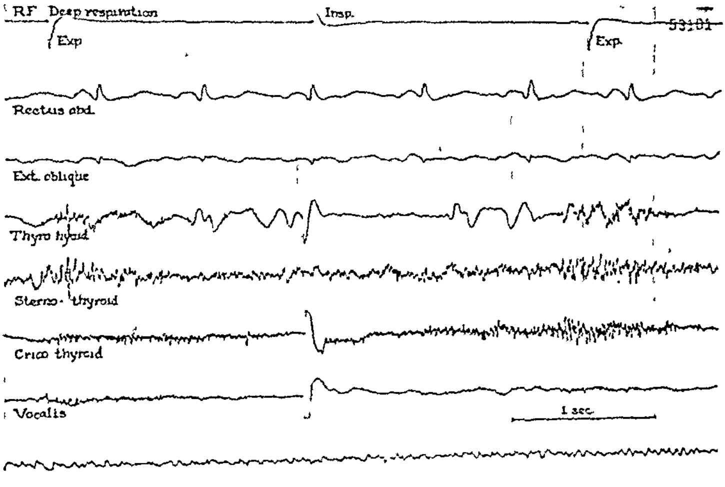

Fig. 2. Electromyograms during deep respiration. Subject R.F. There is considerable activity in the sternothyroid throughout both inspiration and expiration. Thyrohyoid activity is marked only during expiration. Note the increased tone in the vocalis during expiration. Motion pictures showed the larynx moving downward with inspiration and upward with expiration.

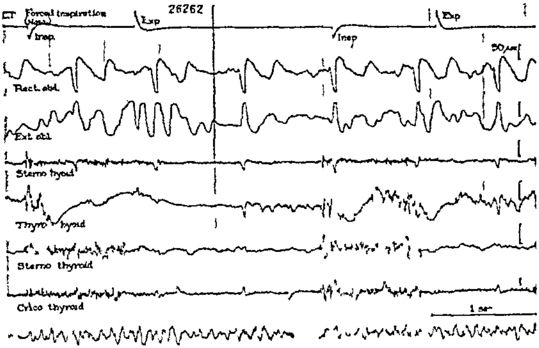

Fig 3 Electromyogram of strong inspiratory effort Subject C T Inspiration is accompanied by increased activity of laryngeal muscles more marked in the sternothyroid than in the thyrohyoid

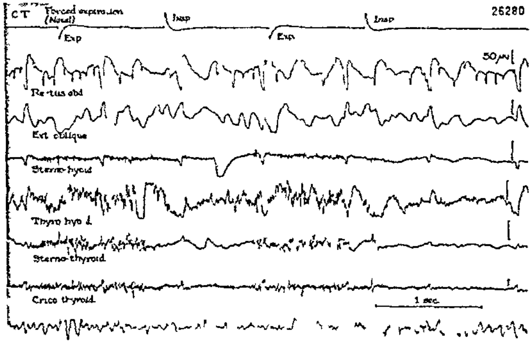

Fig 4 Electromyogram of strong expiratory effort Subject C T Expiration is accompanied by a marked increase in thyrohyoid activity

ference between the inspiratory and expiratory patterns becomes visible. During inspiration sternothyroid activity is well marked whereas the thyrohyoid is relatively quiescent. With expiration there is increased activity in both muscles, but the increase appears most pronounced in the thyrohyoid; there is also a rise in cricothyroid and vocalis activity.

In forced respiratory movements there is increased tone in all the muscles in both phases of breathing; however, in forced inspiration (see Fig. 3) the increased activity is more marked in the sternothyroid than in the thyrohyoid muscle. Motion pictures show that at this time the larynx was moving down (caudad). The rectus abdominus and external oblique are quiescent. With deep expiration (see Fig. 4) there is a striking increase in thyrohyoid potentials, and activity is also registered in the abdominal muscles. At this time the larynx was moving upward.

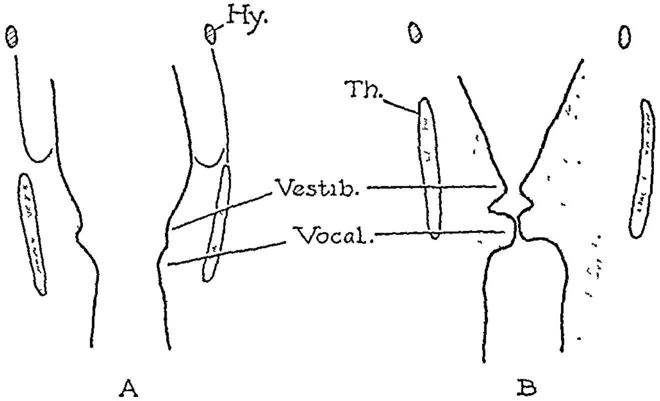

Fig. 5. Anteroposterior laminograms of larynx. Subject R.F. (A) Deep inspiration (B) Expiration. During inspiration there is wide separation of the vocal and vestibular folds. The thyroid cartilage and hyoid bone are also widely separated. In expiration both distances are smaller

Roentgenograms: The anteroposterior laminograms showed that there is wide separation of the vocal and vestibular folds in inspiration (see Fig. 5A); in expiration (see Fig. 5B) the folds were nearer the midline. The lateral Roentgenograms

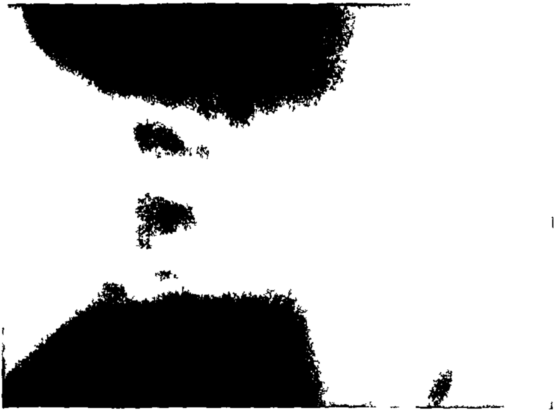

Fig 5B

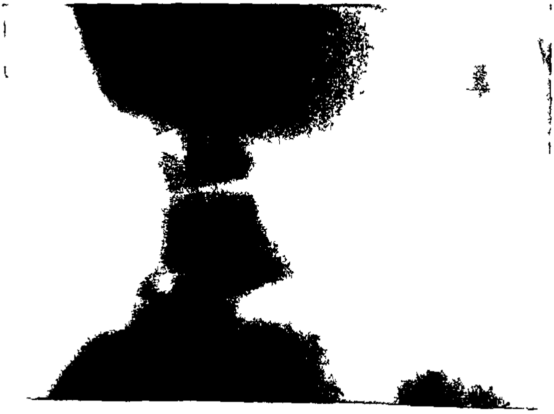

Fig 5A

demonstrated that in inspiration (see Fig. 6A) the larynx moves downward away from the hyoid bone; the anteroposterior diameter of the aditus and vestibule was increased by elongation and forward displacement of the pre-epiglottic body and by backward tilting of the arytenoids. In expiration (see Fig. 6B) the reverse change occurred; the entire larynx moved toward the hyoid, the pre-epiglottic body appeared shortened and thicker and displaced backward, resulting in anteroposterior narrowing of the vestibule.

In expiration the ventricle of the larynx was horizontal and narrow. *At the end of deep inspiration the ventricle sloped* upward and backward and was increased in width.

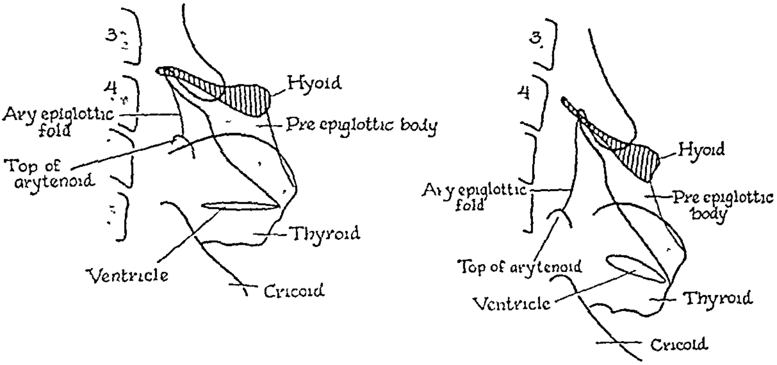

Fig. 6. Lateral roentgenograms of larynx. Subject R. F. Right: Deep inspiration. Left: Expiration. The thyrohyoid distance is greater in inspiration. The lengthening of the aryepiglottic folds and elongation of the pre-epiglottic body are well seen. *The arytenoid eminence, visible at* the lower end of the aryepiglottic fold, is more posterior in deep inspiration. Note the widened ventricle shadow.

Motion Pictures of the Vocal Cords: These showed a striking and constant sequence of events during opening and closure of the glottis. The records obtained by us at 16 frames per second are in agreement with those of the high speed motion picture of the human vocal cords at 1,000 frames per second made by the Bell Telephone Company, through whose courtesy Fig. 7 is reproduced. In the open glottis during quiet respiration the vocal processes were almost parallel to each other and

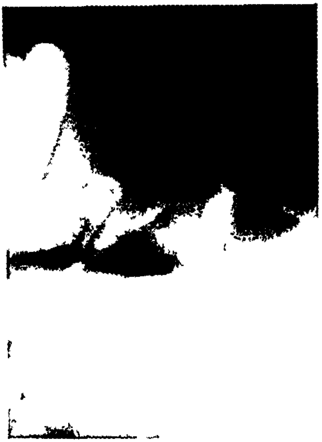

Fig 6B

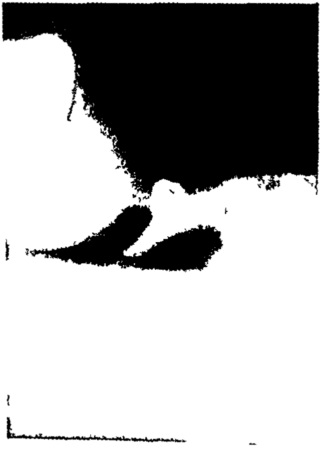

Fig 6A

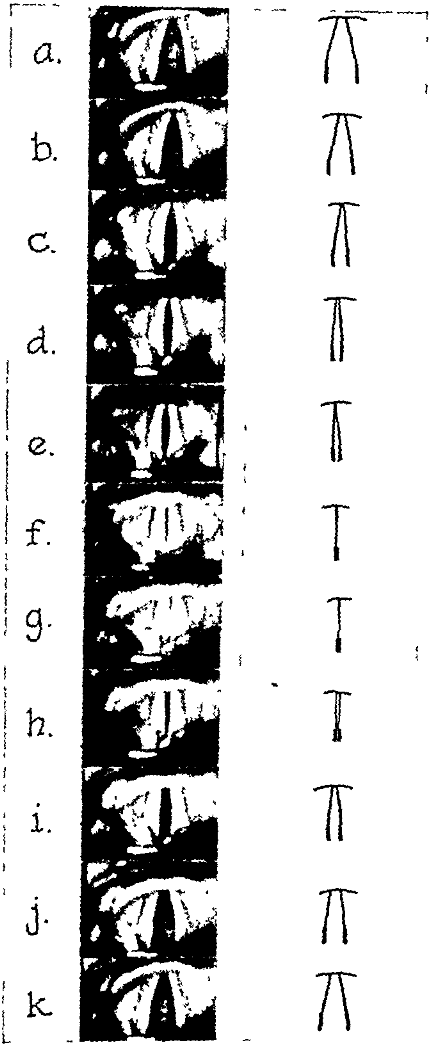

Fig. 7. Motion picture of glottic movements at the start (A-F) and end (G K) of phonation. The vocal processes (indicated by heavy lines in the sketches) retain a virtually constant direction throughout. The interval between frames is approximately 1/40 second (Bell Telephone).

pointed directly forward (see Fig. 7A). As the glottis closed the vocal processes came together but maintained a constant forward direction throughout (see Fig. 7-B to F); there was no sign of internal rotation. When the glottis opened the vocal processes moved apart but retained the same forward direction (see Fig. 7G to J) until just before the maximum enlargement of the lumen (see Fig. 8A to C). The vocal processes then began to diverge, forming an angle of 30° (see Fig. 8D). At the same time the arytenoids were seen to tilt backward and the anteroposterior diameter of the vestibule became enlarged.

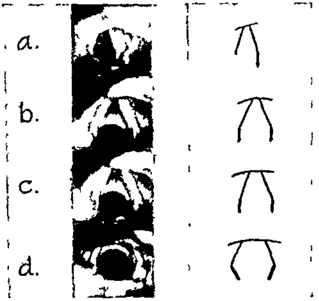

Fig 8 Opening of glottis during deep inspiration (A-B-C) The vocal processes remain parallel (D) At the end of the lateral excursion the vocal processes diverge through an angle of 30°.

DISCUSSION.

The motion pictures of vocal cord movement clearly indicate that changes in size of the glottis are accompanied by sideway sliding movements of the arytenoid cartilages. There is no suggestion of internal rotation at any time, nor of external rotation until the maximum enlargement of the glottis is approached, at which point the vocal processes appear to diverge and the arytenoids tilt backward. This occurs in deep inspiration at a time when the ventricle slopes upward and backward (see Fig. 6A). The divergence is not due to external rotation but is a result of backward tilt of the arytenoids; when seen from above, foreshortening gives this an illusory appearance of rotation. The change in direction of the ventricle ind ess, has moved upward. Since the thyroid and cricoid cartilages have not moved relative to each other, the movement of the vocal process must have occurred by backward tilting of the arytenoid at the crico-arytenoid joint. This joint is cylindrical,[3] with the long axis sloping down at an angle of 45° to the horizontal (see Fig. 9). Backward tilt of the arytenoid around this axis must, therefore, carry the vocal process not

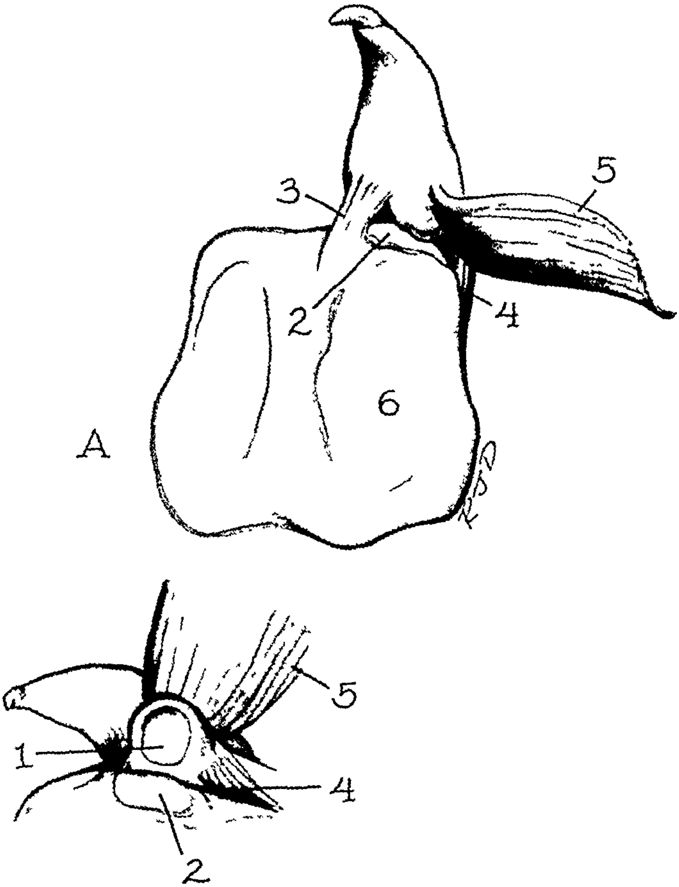

Fig 9. Right crico-arytenoid joint of a man Note the slope and elongation of the cricoid facet (2) and the corresponding concavity of the arytenoid facet (1) The attachments of the posterior crico-arytenoid (5) and lateral crico-arytenoid (4) do not provide leverage for rotation.

only upward but also outward.[4] Seen from above, the outward component appears as divergence. No external rotation is involved.

The motion pictures and X-ray evidence afford no support for the idea that the glottic opening is governed by rotation of the vocal processes; changes are almost entirely due to lateral sliding movements of the arytenoids along the crico-arytenoid joints. This conclusion is supported by observations on three fresh cadaver specimens. In preparations consisting of the hyoid bone, larynx and upper part of the trachea, distraction of the specimens by pulling the trachea and pre-epiglottic body in opposite directions caused separation of the arytenoids and enlargement of the glottis similar to that seen in the motion pictures. Rotation of the vocal processes did not occur. On release of the traction the vocal cords returned to the cadaveric position.

Recognition of the mediolateral gliding motion of the arytenoid during opening of the glottis leads to the question, what muscle or muscles are responsible for this movement? It must be pointed out that in man the muscles attached to the arytenoid cartilages, including the posterior crico-arytenoids, do not originate lateral to the arytenoids and, therefore, cannot by themselves produce a lateral sliding movement of these cartilages. The means by which the arytenoids are drawn apart, therefore, require further consideration.

Signs of the mechanism involved
eral Roentgenograms. In Fig. 6A i
epiglottic distance is increased by t
the larynx and that the upper bord
upward; this indicates that the ve
the border has been pulled upward.
tinue into the aryepiglottic folds
noids upward and outward to the si
the aryepiglottic folds are stretch
larynx, they must exert an upward
arytenoid cartilages and vestibular
tibular folds arch upward and thus
laterally, carrying the vestibular and

the midline (see Fig. 10). When the end of the lateral excursion of the arytenoids is reached the same upward and outward pull apparently makes the vocal processes tilt up and out, causing the body of the arytenoid to incline backward and enlarging both the anteroposterior diameter of the vestibule and transverse diameter of the glottis. In addition descent of the larynx causes stretching of the pre-epiglottic body in the anterior wall of the vestibule, making it lie more vertically and adding to the expansion of the anteroposterior diameter during inspiration.

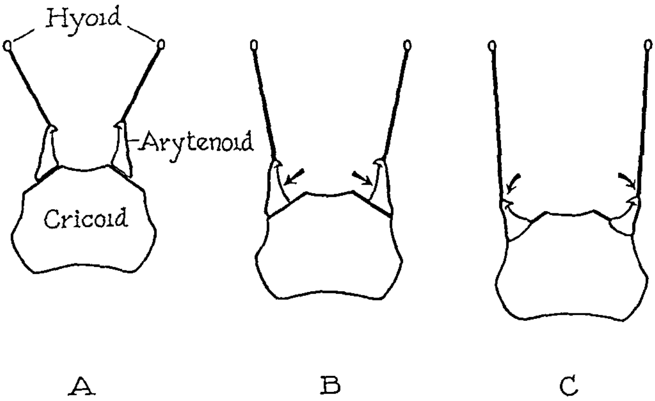

Fig 10 Diagram to show the effect of stretching the aryepiglottic folds on movements of the arytenoid cartilages (A) Expiration (B) Inspiration (C) Maximum inspiration. In inspiration the larynx moves down, away from the hyoid, the aryepiglottic folds become stretched and pull the arytenoids outward In extreme inspiration the stretch acts on the vocal processes (not visible in the diagram) and tilts the arytenoid backward

According to the electromyograms, the sternothyroid muscle is at peak activity during the inspiratory phase of respiration, just when the lumen of the larynx is enlarging in all directions. The action of this muscle tends to pull the larynx away from the hyoid with consequent stretching of the aryepiglottic folds and separation of the arytenoid cartilages (see Fig. 10). Since this action occurs with each respiration it seems reasonable to regard the sternothyroid muscle as playing a major part in opening the glottis and larynx during inspiration. Similarly the thyrohyoid muscle, at peak activity during expiration, pulls the larynx toward the hyoid bone and reduces the stretch on the soft tissues, thus allowing or causing them to fold and approach each other. The thyrohyoid is, therefore, considered to play an important part in narrowing the glottis during expiration.

According to these findings respiratory movements of the larynx and glottis are produced by a mechanism comparable to a bellows. The folds of the bellows are the vestibular and vocal folds (see Fig. 11). When the bellows opens the folds are stretched, and the space between them is increased. In the larynx such a stretch is produced during inspiration by downward movement of the larynx. Conversely, when the bellows closes the folds are nearer together, and the lumen is reduced; in the larynx this occurs during expiration when the organ moves upward. Opening and closure of the glottis during respiration is, therefore, interpreted to be the result of up-and-down movements of the larynx as a whole, controlled by extrinsic muscles, especially the sternothyroid and thyrohyoid. Downward movement of the larynx, produced by the sternothyroid, pulls the larynx away from the hyoid and tends to unfold the vestibular and vocal folds, thereby widening the glottis. On the other hand, the thyrohyoid draws the larynx and hyoid together, causing folding of the laryngeal walls and tending to narrow the glottis. This explains the observations that deeper breathing is accompanied by an increased vertical excursion of the larynx[5] and by a wider opening of the glottis.[6]

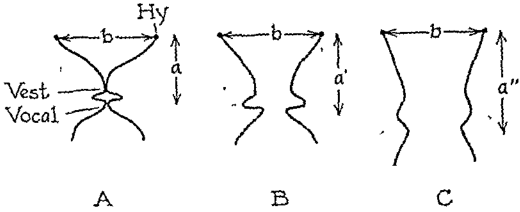

Fig 11 Tracings of anteroposterior laminograms of larynx Subject T T (A) Closed (B) Expiration (C) Inspiration A bellows type of action is exerted on the vocal and vestibular folds by vertical movements of the larynx As distance a a', a", increases the larynx opens at the level of the folds Diameter b remains constant throughout

The role of the crico-arytenoid muscles in glottic movements though essential is probably more indirect than usually supposed, namely to support the vocal process by stabilizing the base of the arytenoid cartilage. The arytenoid cartilage resembles a triangular pyramid resting on one angle of its base at the crico-arytenoid joint (see Fig. 12A); the crico-arytenoid muscles inserted at the joint can be regarded as braces whose function is to stabilize this precarious balance. The forward pull of the vocalis is opposed by the posterior crico-arytenoid, but this creates a torsion moment or torque which it seems the function of the lateral crico-arytenoid to counteract. Such an interpretation explains the continuous activity found in electromyograms of the human vocalis and cricothyroid muscles. It may be surmised that there is a balancing continuous activity in their opponents; *i.e.*, in the crico-arytenoids.

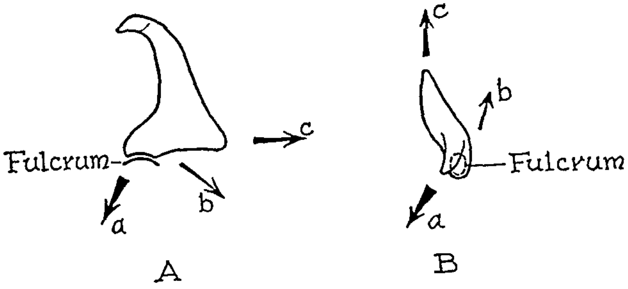

Fig. 12. Forces acting on the base of the arytenoid cartilage. (a) Posterior crico-arytenoid. (b) Lateral crico-arytenoid. (c) Vocalis. The thickness of the arrows is roughly proportional to the size of the corresponding muscles. The fulcrum of any resultant movement is at the posterolateral corner of the base. The forces appear better adapted to controlling anteroposterior tilt than transverse rotation.

The work of Murtagh and Campbell[7] indicates that continuous tone is present in both groups of muscles in goats. It may be concluded that stability of the arytenoid cartilages probably requires continuous tonic activity in all the intrinsic muscles of the larynx, and that it is upon this background that the bellows mechanism operated by extrinsic muscles is superimposed in man. The relation of these findings to the manifestations of recurrent laryngeal nerve paralysis is being investigated.

SUMMARY.

Motion pictures of human vocal cord movements show that opening and closure of the glottis are accompanied by sliding movements of the arytenoids to and from the midline. Rotation of the cartilages does not occur.

Roentgenographic evidence demonstrates that the rhythmic movement of the larynx down and up the neck with respiration causes alternate folding and unfolding of the laryngeal soft tissues, after the fashion of a bellows, and impresses a mediolateral sliding motion on the arytenoids.

Electromyography of intrinsic and extrinsic laryngeal muscles reveals the presence of continuous tonic activity in the intrinsic muscles. It is suggested that continuous balanced activity in these muscles is necessary to stabilize the cricoarytenoid joint. The pattern of activity in the extrinsic muscles indicates that these play a major role in controlling the lateral excursion of the arytenoid cartilages and glottis.

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RECENT ADVANCES IN DIAGNOSIS AND TREATMENT OF MIDDLE EAR DEAFNESS.*†

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During the last few years we have been interested in the treatment of middle ear deafness by prosthetic appliances.[2,3] It was found that some patients may have a good improvement of the hearing function when a prosthesis is used in the tympanum; however, about one-half of the patients do not benefit from a prosthesis. While the successfully fitted patients represent a satisfying and happy group, the unsuccessful case is more thought-provoking and more challenging.

The present study was prompted to analyze the unsuccessful prosthesis patient.

The problem can be formulated as follows: The attempt is being made to improve hearing in non-otosclerotic sound conduction lesions. If a prosthesis is not helpful it is assumed that there exists more than one pathological factor within the sound conduction system. For example, we believe if a patient has an uncomplicated eardrum perforation a closure of the perforation would overcome the functional loss. If the audiometric curve does not change, we assume a second, hidden functional defect which prevents the improvement of hearing. In order to analyze and to find this hidden focus, the otologist must possess a high degree of knowledge of the function in the middle ear.

The problem is considerably more complicated than in otosclerosis. Otosclerosis is simple, even monotonous, insofar as analytical work-up of the function is concerned.

The lesion, so far as the functional aspect is concerned, is always in the area of the stapes footplate. The *tissue* pathology of otosclerosis is osteospongiosis, while the *functional*

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pathology is stapes ankylosis. The tissue pathology cannot be diagnosed clearly in life; however, the functional diagnosis of stapes ankylosis is readily possible. The otologist in dealing with otosclerosis is accustomed to treat the functional entity of stapes ankylosis nearly synonymously with otosclerosis.

There are two principles which have guided the surgical treatment of stapes ankylosis. Speaking in military terms, the ankylosis of the footplate represents a roadblock in the path of sound conduction. There are two ways in military tactics to overcome a roadblock: one means is to circumvent it; this is the idea of fenestration. Second, it can be decided to ram right through the roadblock; this is the concept of mobilization.

For some reasons, which are not always clear or valid, adhesions in the middle ear and their consecutive hearing impairment have been considered unavoidable, unpreventable and unalterable.

The point is, this opinion no longer is unchallenged, and serious attempts are being made to improve non-otosclerotic sound conduction losses.

When fenestration was developed, several stages had to be transgressed:

First, the initial step was the basic physiological recognition that an opening in the vestibular part of the inner ear can be helpful in stapes ankylosis.

The second step was the selection of the site, size and shape of the surgical fenestra.

The third step was the solution of maintaining the opening.

Finally, the audiometric problem of selecting patients and predicting results had to be studied.

We can learn much from the historical steps in fenestration development. It does not take too much imagination to predict that the development of non-otosclerotic surgery has to go through similar steps. Fenestration surgery has already accomplished the training of the otologist by teaching him to operate by microscopic enlargement. Second the dental drill; and last, but not least, the much greater care, exactitude and alertness in every step of the operation. The modern oto-surgeon has gone far beyond the crude and simple technique of "incision and drainage" of the simple mastoid operation.

The surgeon must be audiologist, physiologist, plastic surgeon and experimenter, if he wants to work in tympanal plastic surgery.

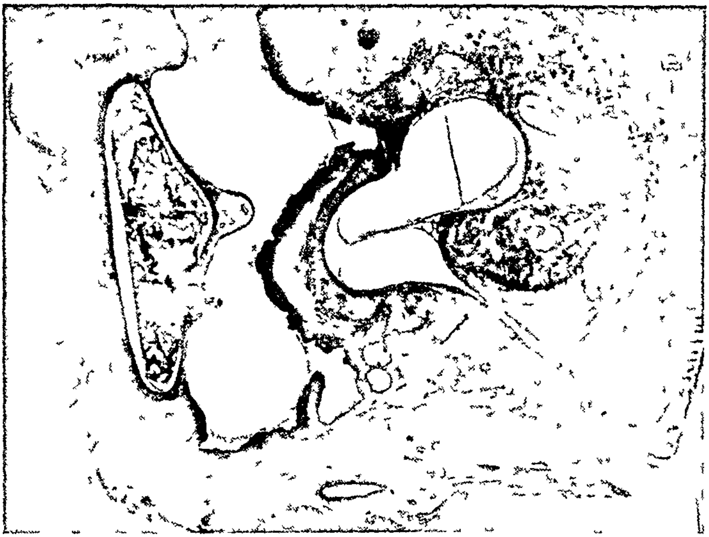

Fig 1 Photomicrograph of the middle ear of patient A B Magnification 8x Vertical section The pathology is characterized by inflammatory and desquamated debris in the external auditory meatus, second, by an eardrum perforation in the inferior half of the pars tensa and swollen mucous membrane on the promontory The inner ear shows no visible histopathology

It is not advantageous that non-otosclerotic surgery be performed first and the selection of patients be learned last. The diagnostic work-up of patients for proper selection of patients must precede the actual surgery. This is the motive for this paper.

There are a number of tests which are helpful in analyzing each individual case. Inasmuch as tympanic plastic procedures are varied and have to be adapted to each individual case, the otologist must command the techniques of these tests, and he must possess the knowledge of physiology to subdivide the middle ear pathology.

Inasmuch as therapeutic procedures in the middle ear may be able to overcome certain features of conduction losses, while failing in others, it seems wise to subdivide the clinical entity "conduction deafness" into several sub-entities which produce components of deafness and must be added together when the final total result of the middle ear deafness is assessed.

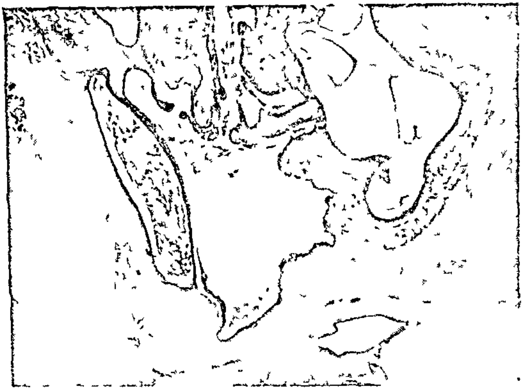

Fig 2 Photomicrograph of the same ear under same magnification as in Fig 1 This section shows extensive adhesive changes in the epitympanum involving the head of the hammer and the body of the incus Connective tissue strands are seen in the oval niche Ring band shows no abnormality

Figs. 1 and 2 show the histology of patient A. B. This patient suffers from extensive middle ear pathology, but shows a normal inner ear. When a hearing test is performed on this patient the sum total of all middle ear defects is recorded. It seems essential to know what percentage of the hearing loss was caused by the accumulation of pus and debris in the outer ear; how many decibels was the signal attenuated on account of the eardrum perforation; to what extent, expressed in decibels, did the conduction of sound in the middle ear suffer from the epitympanic adhesions, and to what extent was the motility of the stapes embarrassed by the swelling of the mucous membrane on the promontory, especially in the area of the oval window niche. Finally, the otologist should analyze the result of any impediment of the phase relation between the two windows. Hypothetically, the course of sound conduction can be mapped in this case about as follows:

The stimulus impinges on the ear with 100 per cent intensity, the plug in the external canal attenuates, *e.g.*, 10 per cent, the remaining 90 per cent sound energy is attenuated again by a further 18 per cent loss due to the perforation of the eardrum. After the two losses of 28 per cent, the remaining intensity is further curtailed by the adhesions in the epitympanum which embarrass the oscillations of the head of the hammer and of the body of the incus. This loss, assumed to be 25 per cent, diminishes the intensity of the signal further. Finally, the pathology of the window area attenuates the intensity further; therefore, finally delivered to the inner ear and transduced into nerve perception, is a message the intensity of which represents the remainder after four or five distinct and locally separated subtractions. The audiogram represents the sum total of these losses. The otologist, if he aspires to the treatment of post-inflammatory, non-otosclerotic conduction deafness, should attempt and should be able to differentiate as completely as possible the parts of which the total hearing impairment was composed.

The following sub-entities should be differentiated:

1. Pathology of the air-eardrum boundary (Drum Pathology).

2. Pathology affecting the sensitivity of the ossicles (Ossicular Pathology).

3. Non-otosclerotic stapes fixation. A disease of the mucous membrane consisting in calcifications of the fenestral area (Stapedial Pathology) (*e. g.*, Tympanal sclerosis of Troeltsch).

4. Pathology in the two window niches (granulations, secretion, tumors, etc.) (Fenestral Pathology).

5. Pathological function of the tube (Tympanal and airspace Pathology).

The following instruments and tests are used for differential diagnosis:

1. Latex rubber to use as artificial eardrum and to determine the functional effect of closure of a perforation.

2. The acoustic probe. This is a point-like sound source which can be applied to various points in the middle ear or on the eardrum. The history of the acoustic probe begins with Pohlman. The probe was a bamboo-like rod which was pushed through a cardboard. The otologist talked against the cardboard and applied the probe to various points in the middle ear. The patient noticed when a yielding spot on the promontory was touched, that the voice of the examiner seemed to increase.

It was an important improvement when Zoellner[5] utilized an audiometric sound source. The advantage is that the tone can be maintained as long as desired. Second, by using attenuation, a measurement in db. can be performed at various intra-otic locations. Intra-otic threshold readings can be taken. We have gone a bit further and have tried to emancipate ourselves from the audiometer and the hearing room and carry our own sound source right with the probe.

3. The tympanal prosthesis is used for elucidation of fenestral pathology. A cotton plug soaked in oil is inserted into the round window niche where it provides sound shadow for the round window; thus, preferential sound conduction is re-established. The obtained hearing gain indicates good function of the opposite window.

4. The Gellé test,[1] now used in quantitative form following the techniques of Thullen.[4]

In 1881 Gellé suggested a specific diagnostic test which was supposed to permit diagnosis of stapes ankylosis. He connected a Politzer balloon with a tube and adapter for airtight insertion into the outer ear canal. When he placed a tuning fork on the balloon the tone could be heard by the patient through the air and the wall of this system. When the balloon was compressed, and thereby the eardrum and ossicular chain

pushed medially, then the loudness of the tone diminished. Gellé interpreted this mechanism as a pressure on the oval window ring band and a proof of stapes mobility. This he named a positive result. The opposite, a negative Gellé test, is the non-appearance of a loudness attenuation for bone conduction on such pressure. Recent re-study and recent improvements of the Gellé test by Thullen make the Gellé test more valuable for the clinician. As a matter of fact, during the decade after the war, most otologists had a hazy recollection of the Gellé test but did not use it. Today, with the Thullen audiometric and thereby quantitative improvements of the Gellé test, the otologist can utilize otherwise not available information.

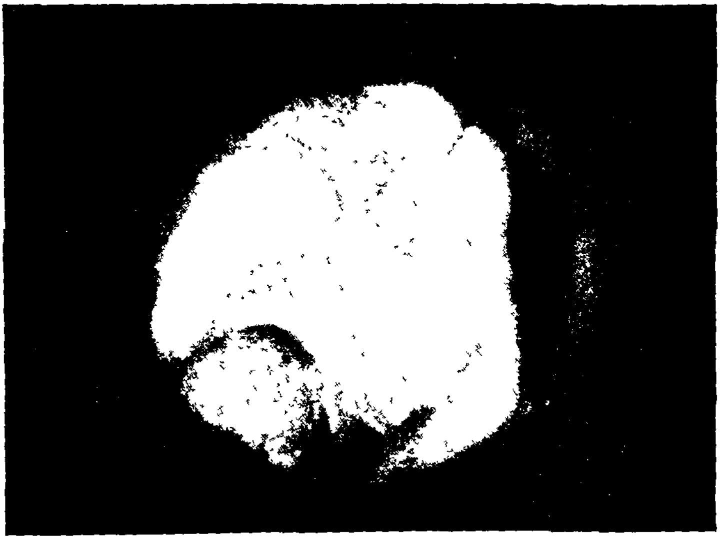

Fig. 3. Black and white copy of a Kodachrome transparency of an eardrum picture. Patient Mrs. F., 45-year-old woman. The pathology of this eardrum is characterized by a round perforation in the inferior posterior quadrant of the pars tensa. Extensive calcareous deposits. When a rubber membrane was placed over the perforation, no hearing improvement occurred. The next step in testing such a prosthesis failure is to test the intactness of the ossicular chain by an acoustic probe. After that blocking the round window niche by an ointment plug should be attempted and the audiological effect of the blocking be noted. Improved hearing after the round window niche has been blocked proves a mobile stapes

The procedure, as advised by Thullen, is executed as follows: A rubber hose with inflatable cuff is inserted into the outer

ear canal. This insures airtight insertion. Coaxially with the tube a tone and a pressure change can be produced. Measurements up to + and − 100 mm. Hg. are possible. The pressure in the outer canal, upon which the patient can no longer hear the tone, is recorded. This pressure is one possible end-point for measurements, or the number of db by which the threshold of audibility changes, can be measured.

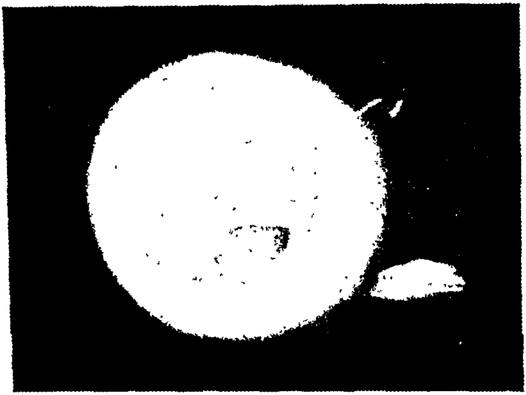

Fig 4. Black and white print of an eardrum Kodachrome transparency. An eardrum with pathology very similar to that of the patient shown in Fig 3 Extensive calcareous plaques in the pars tensa. Perforation of similar size and location as in Fig. 3. Functionally, however, a completely different result was obtained. Closing the perforation increased hearing considerably. The comparison of these two cases demonstrates that the inspection of the eardrum is inadequate to analyze the middle ear pathology. Functional tests are able to furnish additional information.

At present, our main interest is focused on those patients in whom a favorable effect of a tympanal prosthesis is anticipated but fails to be achieved. What physiological condition exists to prevent the principle on which the tympanal prosthesis acts? The principle is the restoration of preferential sound conduction to the oval window. The tympanal prosthesis prevents the simultaneous stimulation of both windows by sound of equal or near-equal phase (see Figs. 3, 4, 5). The logical assumption is that the oval window is not sensitive enough to execute the enforced vibrations; therefore, it is recommended

pushed medially, then the loudness of the tone diminished. Gellé interpreted this mechanism as a pressure on the oval window ring band and a proof of stapes mobility. This he named a positive result. The opposite, a negative Gellé test, is the non-appearance of a loudness attenuation for bone conduction on such pressure. Recent re-study and recent improvements of the Gellé test by Thullen make the Gellé test more valuable for the clinician. As a matter of fact, during the decade after the war, most otologists had a hazy recollection of the Gellé test but did not use it. Today, with the Thullen audiometric and thereby quantitative improvements of the Gellé test, the otologist can utilize otherwise not available information.

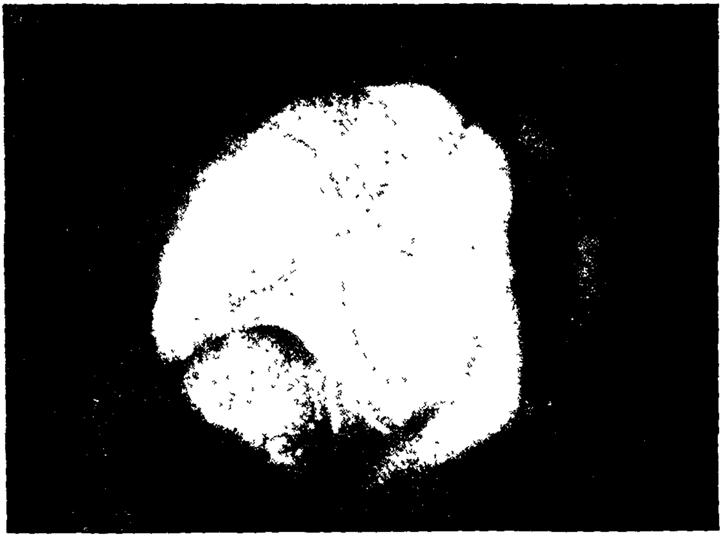

Fig. 3. Black and white copy of a Kodachrome transparency of an eardrum picture. Patient Mrs. F., 45-year-old woman. The pathology of this eardrum is characterized by a round perforation in the inferior posterior quadrant of the pars tensa. Extensive calcareous deposits. When a rubber membrane was placed over the perforation, no hearing improvement occurred. The next step in testing such a prosthesis failure is to test the intactness of the ossicular chain by an acoustic probe. After that blocking the round window niche by an ointment plug should be attempted and the audiological effect of the blocking be noted. Improved hearing after the round window niche has been blocked proves a mobile stapes.

The procedure, as advised by Thullen, is executed as follows: A rubber hose with inflatable cuff is inserted into the outer

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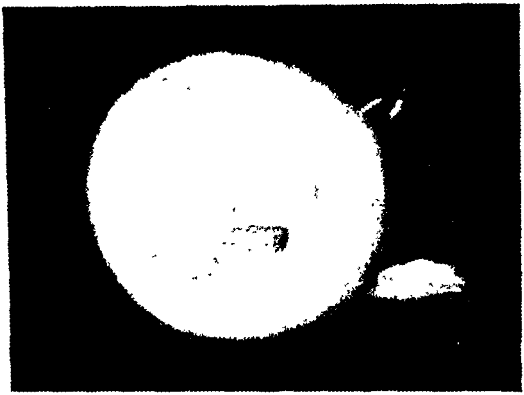

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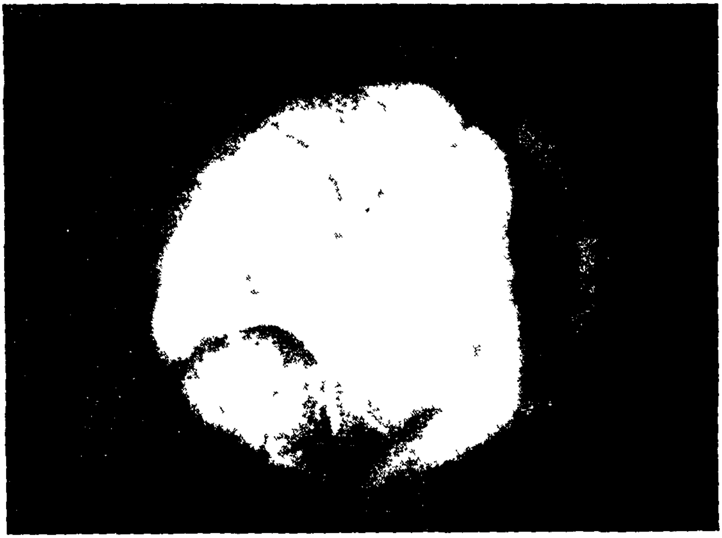

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SUMMARY.

1. Surgical procedures for accomplishing improved hearing function have been developed during the last few years and more are to be anticipated.

2. As the experience with the selection of fenestration patients has shown, careful audiometric and physiological tests must precede the surgery in order to insure proper selection of patient and procedure.

3. The pathology of non-otosclerotic conduction type deafness is more varied as to site, extent and tissue pathology than otosclerosis. It must be recognized that otosclerotic stapes fixation is rather monotonous. There is always a loss of elasticity of the annular ligament; therefore, site and tissue pathology is constant. Similarly, the idea of overcoming this pathology is monotonous. Bypass the oval window as in fenestration surgery or ram through it like in mobilization. Because of this simplicity the surgical treatment of otosclerosis did not demand any great knowledge of the pathological function of the middle ear.

4. In contradistinction, cases of non-otosclerotic middle ear deafness are much more varied as to their underlying tissue pathology, the locations in the middle ear, and the patho-physiological principles by which they are caused.

5. It is suggested that we recognize the following sub-entities of middle ear deafness:

that we test the oval window by touching it with the electronic Zoellner probe. It touching the oval window niche reveals a strong response, a sensitive oval niche is assumed. It is advised to go back to the tympanal prosthesis and block the oval window. We deal now with a system in which the round window may become the port of entry for the sound, and the oval window may be sealed off by the sound shadow effect. Some patients have benefited from oval window fittings.

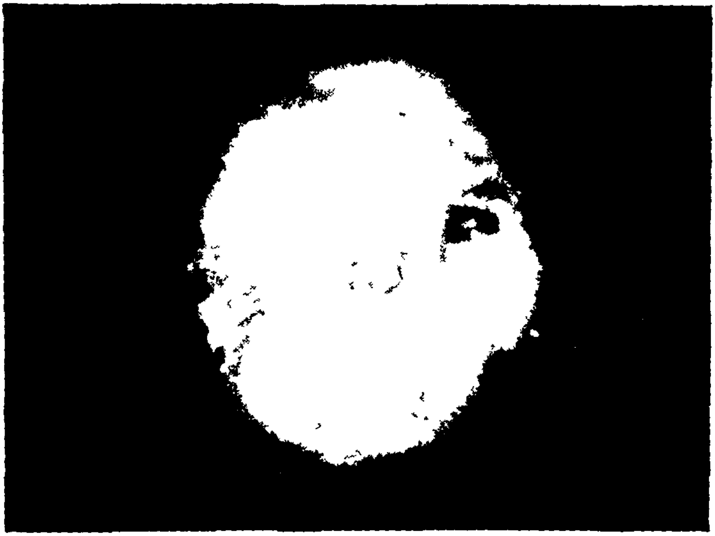

Fig 5 A black and white print of an eardrum Kodachrome portrait The perforation in the posterior aspect of the pars tensa has about the same size as in Fig 3 and Fig 4 In this case nature did a tympano-stapedo-pexie There was good hearing considering the extensive pathology It is likely that the ossicular chain was interrupted but a columella effect established by the adhesion of the stapes head onto the pars tensa

In case the oval window is found insensitive, surgical cleansing of the oval window niche may be indicated.

It is not advisable, according to Zoellner, to probe and to manipulate too much in the round window niche.

Another reason for failure of the tympanal prosthesis is cystic degeneration of the promontorial wall.

A Politzer maneuver is indicated and advised as the next step. The reason is that there may be cystic changes or adhesive attachments of the drum membrane onto the promontory adjacent to and including the niche of the round window. When the Eustachian tube can be successfully inflated a ballooning of the adherent eardrum may result. A maintained although reduced airspace, comprising the tubal ostium, the hypotympanum and round window area is created. This reduced airspace has been named "the small tympanum", and its creation by Politzer inflation is functionally improving and diagnostically and prognostically significant. It can be produced surgically by a skin flap which covers the hypotympanum, the tubal ostium and the round window.

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5. It is suggested that we recognize the following sub-entities of middle ear deafness:

a. Diseases of the eardrum (Functional pathology of the air-eardrum boundary).

b. Diseases of the ossicular chain (Ossicular pathology).

c. Diseases of the stapes (Stapes pathology).

d. Diseases of the windows (Fenestral pathology).

e. Diseases of the tube and tympanal airspaces (Airspace pathology).

6. Finally, the selection of surgical procedures should be based on a knowledge of the individual factors in each patient. The following treatment procedures are being developed:

a. Closure of an eardrum perforation by plastic flap.

b. Freeing epitympanal structures from scars or sclerotic membranes.

c. Freeing the oval window from obstructing tissues like granulations, scars and membranous sclerosis (Zoellner).

d. Tympano-stapedo-pexie. This method mentioned already by Stacke and convincingly shown in this country by Juers.

e. Creation of a columella (Wullstein).

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CHANGING CONCEPTS IN AUDIOLOGY.

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Audiology, as presently conceived by many, began primarily after World War II, and since that time there has been a new emphasis and interest in the problems of people with impaired hearing. This new emphasis was given substantial impetus by the rehabilitative needs of hearing impaired veterans. While this has been advantageous, certain problems have arisen as a result. One of the most generally accepted functions of an audiology center became that of dealing with amplification methods and procedures. The hearing aid became a focus of interest and perhaps was uncritically emphasized as a panacea for the hearing impaired individual. In any event, most programs gave considerable attention and study to the question: "What is a good hearing aid and to whom should it be fitted?" To a certain extent this meant that audiological centers became hearing aid evaluation centers. Although such programs brought benefits to many people and fostered advancement of knowledge, it is now apparent that they had limitations, both theoretically and clinically.

CONCEPTS ARE CHANGING.

During recent years audiological centers have changed their emphasis from major concern with hearing aids to broader aspects of auditory disorders. The significance of an auditory impairment now is viewed as being pervasive and inclusive. Other aspects of the problem of deafness are being emphasized; the concept of audiology is changing. Audiology is not limited to the problem of peripheral deafness; that is, deafness which is the result of deficiencies of the hearing mechanism. Currently audiology emphasizes the more inclusive concept of auditory disorders and auditory behavior. It is concerned with the total problem of inability to receive and to profit normally from sound, irrespective of the nature of the pathology involved. At the broadest level this means that

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audiology is a science which is concerned basically with the receptive phase of language development and acquisition. It deals with all aspects of hearing but is fundamentally concerned with the reception and comprehension of spoken language. The new concept emphasizes all types of auditory disorders, disturbances of auditory behavior, the pathology of hearing, as well as the study of normal hearing and its significance to the human being.

TYPES OF AUDITORY DISORDERS.

Deafness.

Impaired hearing acuity in infancy impedes the reception of speech and thereby disturbs a basic maturational factor in human development, the acquisition of verbal symbols called speech. The effect of this deprivation is pervasive and influences the total behavior of the individual. Likewise, a hearing impairment which occurs in later life causes the individual considerable distress through isolation, increased anxiety and in other ways. These problems continue to receive profound consideration and study, not only from the point of view of the nature of deafness but also relative to its meaning and impact on the individual. As concepts change, however, it is necessary to emphasize other auditory disorders which affect reception of sound.

Disturbances of Auditory Perception.

Auditory perception is the way in which the human being organizes, structures and uses the sounds in his environment. In order for the individual to profit normally from auditory experience he must have normal auditory perceptual capacities. Auditory perception includes the constant awareness of all sounds in the environment, and the ability to select the appropriate sounds from this conglomerate world of sound. All sounds in the environment are not equally useful simultaneously. Those which are not useful at a given moment must be held in the background of attention while those which are immediately relevant must be brought into the foreground.

A revealing example of this process is seen when an individual is listening intently; he is deliberately attempting to make a particular sound come into the foreground of attention. In order to do so he simultaneously attempts to keep all other sounds in the background. This is referred to as *listening behavior*. It is characteristic of children who have disturbances in auditory perception to be able to hear but to be unable to listen. They cannot respond to their auditory world selectively and meaningfully; they cannot structure their auditory world. As a result they respond to all sounds as though they are of equal importance simultaneously. Such children often are seriously distractive, disinhibited and unable to give sustained attention. Because this is a basic threat to the adjustment, if not to the survival of the organism, some of these children suspend their use of hearing and intermittently appear to be deaf. Such confusion of auditory perceptual incapacities with deafness is enhanced by the fact that typical tests of hearing assume auditory perceptual integrity. In other words, most tests of hearing assume ability to listen. Because individuals who have disturbances of auditory perception cannot listen, they will be erroneously classified as hearing impaired if such test results are interpreted literally.

Another significant factor audiologically is the relationship of auditory perception to the use of hearing aids. Perhaps one of the most difficult aspects of the use of a hearing aid is the manner in which it affects the individual's ability to structure the world of sound. Apparently the hearing aid alters the normal auditory perceptual processes in a critical manner. The hearing aid user seems to be confronted with a difficult auditory perceptual task which has been studied only superficially. Presently the typical hearing aid or auditory training unit makes it possible for the user to function only in a foreground manner. This can be considered as a basic reason for the user's not being able to understand speech in a noisy environment or when he is in a group conversational situation.

Through a hearing aid the fundamental process of separating the world of sound into foreground, background, important and unimportant is grossly disturbed. The ways in which hearing aids affect auditory perception is urgently in need of scientific study. Perhaps the new hearing aid equipment using two microphones, and which makes depth perception (foreground-background) possible will bring untold benefits to the hearing impaired during the next few years.

Receptive Aphasia.

Another disorder which must be considered comparatively with deafness is receptive aphasia. This differs from deafness in that it is not an inability to hear but a deficiency in the interpretation of the spoken word. Like disturbances of auditory perception it is due to brain injury, but it differs from an auditory perceptual disturbance in that it is a symbolic disorder. An individual with an auditory perceptual disorder is capable of interpretation if he can organize sounds and select them appropriately from his environment. This is not true of the receptive aphasic. His basic incapacity is not one of structuring his world of sound (many receptive aphasics also are disturbed in auditory perception), but of relating the spoken word to experience. He cannot understand the spoken language symbols (speech) which normally represent the world of objects, feelings and ideas. Receptive aphasia is more common in children than has been assumed. Concepts of audiology include the need for differential diagnosis and management, both medically and nonmedically, of deafness versus receptive aphasia.

Psychic and Psychogenic Deafness.

Emotional disturbances might impede the normal reception and use of sound. Audiology has emphasized the importance of psychogenic deafness in adults, but only recently has stressed the importance of psychic deafness in children. Clinical experience and research continue to reveal that if sound becomes seriously threatening and unduly anxiety-producing, the organism suspends normal use of hearing to prevent disintegration.

For both theoretical and humanitarian reasons this is a provocative and necessary aspect of an inclusive concept in audiology. Hearing is a fundamental distance sense through which the human being acquires a unique human characteristic, the ability to speak. For this important function alone hearing has marked significance psychologically; but this is not all that the individual receives through hearing; through it he gains feelings of well-being, identification with others, esthetic experiences, a non-directional "antennae" contact with the happenings in his environment, signals of danger and many other types of experiences such as expressions of hate, taboos, rejection, threat, punishment, hostility and fear. If one's auditory experiences generate primarily disequilibrium with the environment, then a fundamental way, even if unsatisfactory, in which to deal with the unbearable stress is to behave as though hearing is not present. Relinquishment of hearing is an indication of serious emotional disorder, and should be treated as such. Psychic and psychogenic deafness are areas requiring further emphasis collaboratively with otologists, psychiatrists, clinical psychologists, pediatricians and audiologists. Currently it appears that the most common cause of psychic deafness in children is childhood schizophrenia. In childhood it seems that such a basic organismic function as hearing typically is not relinquished for psychological reasons unless the emotional breakdown is severe and a psychosis is present. This may be in contrast with psychogenic deafness in adults, which in general seems to manifest neurotic rather than psychotic conditions.

Central Deafness.

Although central deafness is not well understood it must be included in the conceptual frame of reference in audiology, because presumably it is a factor in the reception and use of sound. Central deafness refers to impairment of the auditory pathways in the brain. This impairment impedes the conduction of the sound impulses to the interpretative areas of the brain but does not include damage to the interpretive areas themselves. (The latter condition results in aphasia.) Audiology, with the assistance of otology, neurology, clinical psychology and other sciences, is gradually making progress in the clarification of this problem with implications for treatment and training for both children and adults who present this type of auditory disorder. Experience with young children suggests that hearing for startle and other reflex behavior may be normal but that bilateral brain damage prevents conduction of sound to higher levels, so that the child is deaf for purposes of language development. This might be one of the reasons that some children who have normal hearing by psychogalvanic audiometry are like deaf, brain-injured children clinically and educationally.

Auditory Agnosia.

Auditory agnosia is understood chiefly as a neurological disorder in adults. Clinical experience with auditory disorders suggests that it also might be important in children. It differs from receptive aphasia, deafness, central deafness and psychic deafness. It is due to gross damage to the auditory interpretative areas in the brain. The receptive aphasic comprehends non-speech sounds, such as the sound of an auto horn and the quacking of a duck. His inability to interpret sound is limited to the spoken word. The auditory agnosic, on the other hand, cannot interpret any sound normally although as compared to the individual with deafness, he can hear. Diagnosis, especially in children, is difficult; however, under careful study these patients usually manifest awareness of sound. Such awareness may be revealed more readily when a faint sound is discontinued rather than when it is introduced.

EDUCATION AND TRAINING.

Speech, Speech Reading and Reading.

Audiological concepts are changing also in regard to the education and training of both children and adults with impaired hearing. There is a new interest in the problems of teaching speech, speech reading, reading and language to the deaf and hard-of-hearing. This new emphasis has been referred to as educational audiology. Perhaps the most significant aspect of this shift in emphasis is that scientific research methodologies are being applied to these areas. Tests are being devised for evaluating speech reading success, to ascertain progress in speech development, and for evaluation of the basic symbolic process used by the deaf child; likewise, his perception, vision and other aspects of development and behavior are being studied in order to better understand the learning problems related to deafness. This includes the awareness that speech reading is a visual symbol system and that speech for the deaf might be essentially a tactile-kinesthetic symbol system. Language teaching might require individualization on the basis of a visual-tactile approach with one individual and an auditory-visual-tactile or a visual-tactile-auditory approach with another. A deliberately planned approach on the basis of individual capacities and needs seems clearly indicated.

Assumptions have been made for techniques and methods of teaching the deaf and hard-of-hearing which have not been objectively evaluated. It is becoming possible to evaluate these assumptions with more scientific accuracy.

Auditory Training.

Auditory training is the process of teaching the individual to use his residual hearing as effectively as possible. This is done through the use of auditory training equipment and through group and individual hearing aids. Several clarifications in the use of auditory training are indicated. The limits of deafness beyond which auditory training is unsuccessful and thus unrealistic have not been well ascertained; furthermore, objectives for gradations, success and usefulness are necessary; there is considerable difference in training a person to be aware of traffic sounds and in training him to comprehend speech sounds. Clarification of the objectives and purposes of auditory training would assist the individual, and the parents, to accept limitations and not over-anticipate outcomes. The changing concepts in audiology include the knowledge that auditory training cannot be used uncritically to the maximum benefit of those with marked hearing impairment.

IMPLICATIONS OF DEAFNESS AND THE MULTIPLE HANDICAPPED.

Audiology has stressed the hearing mechanism and the use of amplification. As the concepts change the effect of deafness is being increasingly emphasized; deep concern has developed regarding the total needs and functioning of the hearing impaired person. Studies include the effect of deafness on mental development, on memory, on personality and adjustment and on motor functioning. The needs of the deaf and hard-of-hearing who are also brain-injured, mentally retard bral palsied or blind, as well as the needs of the aged with presbycusic deafness, are receiving increasing consideration. Such work is being accomplished with the collaboration of various allied professional groups. As this work progresses it is apparent that audiology has a responsibility to encourage counseling and programs of psychotherapy for those with auditory disorders and for their families. Such services await development in most respects.

SUMMARY.

The following are indications that the concepts in audiology are changing:

1. The field is becoming more inclusive and emphasizes all auditory disorders which affect language reception.

2. Diagnostically the frame of reference is that of auditory disorders; it is not simply testing hearing but it is also the differential diagnosis of individuals with disturbances of auditory behavior.

3. Therapeutically it is not only the fitting of hearing aids, prescribing auditory training and speech reading but is also the outlining of individualized, inclusive educational and rehabilitative programs.

4. The patient is not viewed as being only deaf or hard-of-hearing, but he is also understood broadly in terms of having sustained a sensory deprivation which affects every facet of his behavior and his relationship to his family and to society.

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AN UNUSUAL CASE OF EOSINOPHILIC GRANULOMA

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Eosinophilic granuloma is a condition characterized by the presence of localized rarefactions involving one or more bones Single lesions are more common in older children and in young adults, as contrasted to multiple lesions which are more common in younger children The onset is usually insidious and symptom free, the lesion being detected only incidentally by X ray and after the occurrence of pathological fractures The symptoms when present include localized pain and tenderness with swelling of the adjacent soft tissues Leucocytosis with moderate eosinophilia may also be present The most common sites of predilection are the bones of the cranium, pelvis and those of both upper and lower extremities, however, any bone may be affected

The X-ray picture shows a clear cut, round or oval lesion with the overlying cortex intact and not expanded unless destroyed by the process

Biopsy reveals the presence of a granulomatous, inflammatory lesion infiltrated by monocytes and polymorphonuclears with varying numbers of eosinophiles

Treatment is surgical excision and Roentgen therapy, followed by rapid cure

Prognosis must be guarded, because of the possibility of lesions appearing elsewhere in the skeletal system as well as in the skin and viscera with transition of the disease into Hand Schuller Christian syndrome or Letterer-Siwe disease

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CASE REPORT.

This is the case of W. R., three-year-old male, from Puerto Rico, who was admitted for the first time at the Martland Medical Center with the chief complaint of painful swelling over the chin.

The present illness began about three weeks before admission, when the patient, while playing with his brother, received a slight blow on the chin and immediately developed pain over the area. The parents noticed swelling of the chin at this time, and the patient was brought to the hospital.

Family history was essentially negative. Past history revealed the usual childhood diseases.

On admission, the patient still complained of pain. Examination revealed slight swelling and tenderness over the region of the symphysis menti. No other contributory findings were noted.

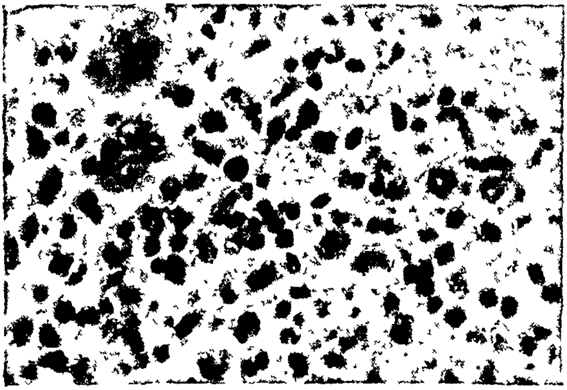

Fig. 1.

Laboratory examinations were performed. The hemogram revealed a *hemoglobin 10.5 grams;* red blood cells 4,350,000; white blood cells 5,050; and a differential with Neutrophils 38 per cent, Lymphocytes 58 per cent, Eosinophiles 1 per cent, Basophiles 1 per cent, and Monocytes 1 per cent. The sedimentation rate was 33 mm./hr. (Westergren) ; cholesterol 143 mg. per cent; calcium 9.7 mg. per cent; alkaline phosphatase 6.8 Bodansky units. Kahn was negative. Urinalysis was within normal limits. X-rays revealed a destructive process involving the anterior portion of the mandible. There was overlying soft tissue swelling. A small amount of subperiosteal bone was seen inferiorly at the anterior aspect of the mandible. The etiology could not be definitely stated from the Roentgen studies alone. X-ray of the skeleton was negative.

A surgical biopsy was performed. There were irregular, small, cystic areas in both mandibular bones in the region of the symphysis menti.

A specimen was removed and submitted to the laboratory.

The pathological report was as follows

Section is characterized by an unusual inflammatory reaction composed of numerous large histiocytes and cells of the early immature granulopoetic series Numerous small and large multinucleated giant cells are present, and many eosinophilic polymorphonuclear leucocytes are scattered throughout Section is very vascular and congested Considerable interstitial hemorrhage is present

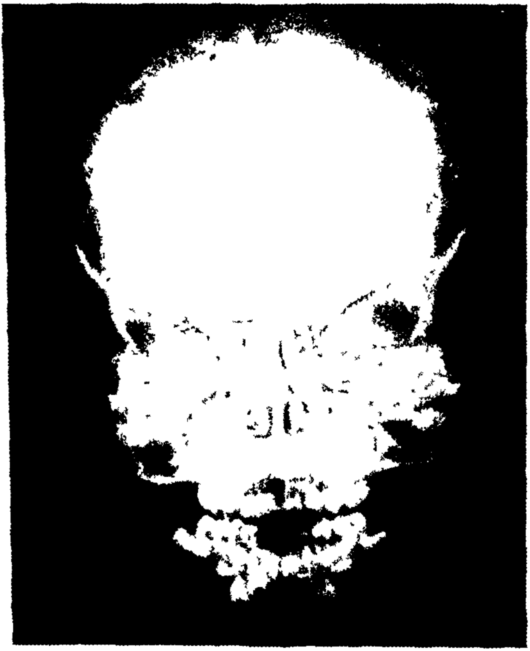

Fig 2

DIAGNOSIS Eosinophilic granuloma

About one week later, the wound from which the biopsy had been obtained broke down, and there was a protrusion of soft reddish vascular tissue from the wound opening, accompanied by pain and tenderness

A large amount of soft tissue was removed, followed by curettage of the surrounding bone. Specimen was submitted to the laboratory for histopathological examination, and the diagnosis confirmed the initial impression of eosinophilic granuloma.

X-ray examinations of the mandible were taken after the operation, and no further progress of the disease was noted.

Sutures were removed on the fifth day, and the wound healed by primary intention.

CONCLUSIONS.

1. The case is considered unusual because of the following:

   a. This is the first case of solitary eosinophilic granuloma of the mandible to be reported. Of all the cases in the medical literature, there are two cases that show mandibular lesions, but other manifestations of eosinophilic granuloma were present in the body at the same time.

   b. A single lesion in a three-year-old child in contrast to the general opinion that multiple lesions of eosinophilic granuloma are more common in young children and the single lesion in older children and young adults.

2. It can' be said that eosinophilic granuloma responds to surgery, as shown in this case.

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MEDICAL OTOLARYNGOLOGY.*

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It is a well-recognized fact that otolaryngology has for many years gradually developed into a greater medical than a surgical specialty. Perhaps the earliest transformation began with the recognition of allergy as an important cause of diseases in the nose and paranasal sinuses. More conservative surgical interference has taken precedence over the radical. More recently the introduction of the antimicrobial agents has led to a greater reduction in the necessity for surgical procedures, especially on the mastoid.

Although otolaryngology has become a predominantly medical specialty the change represents great advancement. The otolaryngologist of today must be an allergist, a bacteriologist, an immunologist, a nutritionist, a psychologist, and he must be familiar with many medical problems which are associated with or related to his specialty.

In an analysis of the data from 1,000 consecutive ear, nose and throat patients observed in 1954 by Davison[1] at the Geisinger Memorial Hospital and Foss Clinic, he noted that if minor operations were excluded, only 20 per cent of the 1,000 patients needed some type of operative procedure. The other 80 per cent required good differential diagnosis, advice concerning living habits, reassurance, minor psychotherapy and drug therapy. In the private practice of otolaryngology in which there is no close association with a hospital or a clinic, the incidence of surgical cases should be much lower than 20 per cent.

In Davison's series of 1,000 cases, some type of allergy was a factor in 22 per cent, and he expressed the opinion that it is not necessary for the rhinologist to refer these patients elsewhere for treatment. About 11 per cent of the group had vestibular disturbances and deafness. Twenty per cent had

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headaches, tension, cancer phobia, carotid pain, globus, and no ear, nose or throat disease. The remaining 27 per cent represented miscellaneous diseases such as external otitis, otitis media and conditions encountered in bronchology, etc.

The problems encountered in medical otolaryngology must be approached primarily with a complete otolaryngologic and general medical history. A history questionnaire of the type recently introduced by Anderson and Rubin[2] for allergic patients may be employed to obtain information which the busy physician does not have time to procure. The local physical and technical examinations should be thorough, including all pertinent laboratory procedures such as the cytologic, bacteriologic, radiologic, pathologic, audiologic, bronchologic, etc.

In those instances in which some closely related medical conditions may be present, as in those patients who have been referred by another physician, the otolaryngologist should extend the laboratory examination to include an X-ray of the chest, a complete blood count and hemoglobin determination, a sedimentation rate, a B. M. R., a urine examination or additional procedures as indicated. The blood pressure should be taken and a physical examination of the chest, if necessary. In many instances the otolaryngologist may establish a tentative diagnosis and thereby he may be able to refer the patient for some specific type of therapy.

The problems of medical otolaryngology are varied, multiple and ever changing. In many instances the indications for surgical interference must be evaluated only after medical treatment and observation. The management of the patient, therefore, is directed to the consideration of disease and loss of function which affect the ears, nose and throat, and related structures. These problems concern allergy, infection, immunity, anemia, nutrition, dental care, effects of tobacco and alcohol, endocrine deficiencies, loss of function as manifested by vascular headaches, vertigo and tinnitus, deafness, psychosomatic disturbances and other related conditions.

The consideration of all the subject matter can be discussed here only briefly. The recent five volume *Encyclopedia of Otolaryngology*, edited by Coates, Schenck and Miller,[3] is a comprehensive treatise which presents detailed discussions by leading authorities on both medical and surgical subjects. Perhaps the most important problems encountered in the practice of otolaryngology today concern chiefly allergy, infection and immunity. More recently their management has been modified to a great extent by the introduction of the hormonal agents (ACTH and Cortisones), the antihistamines, the antibiotics, vaccines, certain pharmacologic agents and more attention to secondary factors.

RESPIRATORY ALLERGY.

The diagnosis and treatment of respiratory allergy[4] must be based upon the analysis of a detailed clinical history, supplemented by the questionnaire as recently presented by Anderson and Rubin, complete local examinations and relevant laboratory procedures. This investigation will determine primarily whether the presenting problems concern nasal allergy and hay fever (with or without sinus involvement), nasal polyps, laryngeal edema, cough and bronchial asthma, external ear, Eustachian tube, middle or internal ear involvement, oral lesions and salivary gland swellings, allergic headache or local dermatitis. Only one, or a combination of these manifestations may occur. In addition, however, other associated allergic lesions may involve the gastrointestinal tract, genitourinary organs, the skin, the nervous system, the joints, the blood, the blood vessels and other structures in the body.

An analysis of this investigation or approach to the presenting problem often suggests the etiologic agents to be considered, such as: house dust, animal danders, and other inhalants as well as pollens, which commonly cause nasal and sinus symptoms, Eustachian tube involvement, cough and bronchial asthma. Foods and drugs are not common primary factors but may be secondary to the inhalants. Foods and drugs may cause middle and internal ear involvement, salivary gland swellings, laryngeal edema, headache and local atopic dermatitis. These lesions may be associated with others located elsewhere in the body; for example, in the gastrointestinal tract and in the skin. Contact allergic lesions may be located in the oral or pharyngeal cavities or on the skin of the auricles, around the mouth and the ocular regions. Other secondary factors which concern psychosomatic, endocrine, nutritional, dental care and other states must be considered also.

Diagnostic skin tests by the scratch and intracutaneous methods may or may not reveal significant information; therefore they cannot be relied upon as the basis for treatment in the majority of cases.

For diagnosis and for determining the clinical course of respiratory allergy, frequent cytologic and bacteriologic examinations of the secretions must be made for eosinophiles, neutrophiles, epithelial cells and bacteria. Superimposed acute infections are common in allergic patients, so routine treatment must be modified frequently in the management of the patient.

Adequate management of the allergic patient begins with the avoidance or elimination of all suspected or potential etiologic factors. Specific treatment may be directed to injection therapy with house dust, other inhalants, molds and pollens, dietary manipulations and adjustment. Symptomatic treatment concerns the use of ACTH, Cortisone, endocrines, drugs such as ephedrine, antihistamines, antitussives and Aminophyllin compounds, etc.

In the markedly acute cases or in those difficult ones in which the routine therapy is not satisfactory, such as severe uncontrolled hay fever, without or with asthma, perennial nasal allergy, nasal and sinus polyposis, acute urticaria and contact atopic dermatitis, ACTH and the Cortisone (Prednisólone) compounds may be administered for a short period of time, usually one to two weeks. In most instances there is a dramatic relief from symptoms, but the good results cannot be maintained after the discontinuance of these hormones until allergic management has been adequate. In some cases, however, it may be necessary to maintain the patient on a small daily dosage for an extended period with no untoward side effects.

We usually begin therapy with the administration of 40 units of ACTH Gel or Corticotropin Z (Organon), along with an initial dose of 30 mg. of prednisolone (Delta Cortef, Meticortelone, Hydeltra, Sterane, etc.) divided into four doses: one dose after each meal and at bedtime. Subsequent daily divided doses are usually reduced as follows to: 25 mg., 20 mg., 15 mg., 10 mg., 5 mg., 2½ mg., or less. In some instances it may be necessary to reduce more slowly at the 15 to 10 mg. levels in order to maintain relief. Finally the lowest or minimum effective dosage is established, and subsequently the use of the hormones may be discontinued. In relapses, 10 to 15 mg. daily may be sufficient. The second dose of ACTH is usually 20 units on the third or fourth day, then 10 units after seven or eight days. When the prednisolone daily dosage is small the use of ACTH is unnecessary. Non-specific stimulation of ACTH secretion may be maintained by the intradermal injection of about 1/10 unit of Staphylococcus toxoid twice weekly, along with other injection therapy.

The most dramatic results with this type of therapy are noted in cases of nasal polyps. They may disappear from the nose completely within 7 to 14 days. Local treatment with a cortisone spray may be used also in some cases. Sustained results, however, depend upon adequate allergic management. This plan of therapy is also effective in bronchial asthma and in hay fever.

Hay Fever: Most patients with ragweed hay fever present a problem of multiple sensitivities such as those to foods, molds and miscellaneous inhalants. Treatment, therefore, concerns the balanced management of all these factors which is usually very difficult in about 25 per cent of the cases. Bronchial asthma usually complicates these cases in the latter half or one-third of the season. In a group of more than 100 of these difficult cases treated during the 1955 ragweed hay fever season, excellent results were obtained in almost 100 per cent by the additional use of ACTH and Delta Cortef (prednisolone) in maintenance doses of 10 to 15 mg. daily. During the 1955 season we prescribed ephedrine sulphate in ⅛ to ¼ gr. doses for symptomatic relief instead of antihistamines, with better results. We feel that the latter drugs have a blocking effect on immunologic therapy.

RECENT ADVANCES IN ALLERGY AND IN INFLAMMATION.

From these observations it is evident that the hormonal agents play an important part in allergic management. Although they do not cure the disease, they apparently attenuate or reduce the degree of sensitivity so that immunologic procedures become more effective.

The recent work of Godlowski[5] on the "Enzymatic Concept of Anaphylaxis and Allergy" sheds new light on the fundamental mechanism of these conditions. His studies concern the cellular enzymatic system, the effect of hormonal agents and the part played by eosinophiles. He demonstrates the close interdependence and causal relationship between disturbed enzymatic proteolysis and hormonal dyscrasia.

On the basis of his studies on isolated cells and exudates, Menkin[6,7] showed that the anti-inflammatory action of ACTH and Cortisone takes place at the cellular level. By suppressing the inflammatory process, certain infections may cause undue harm, but in other types of inflammation caused by non-viable organisms, such as ophthalmologic disorders or in allergic processes, these anti-inflammatory reactions may be of great value.

Menkin[6,7] points out further that inflammation is first manifested by an increase in capillary permeability and the release by the injured cells of leukotaxin, a polypeptide-like substance, which also induces the local migration of polymorphonuclear leukocytes. Inflammation is, therefore, the regulation of bacterial invasiveness. Menkin also showed that the injured cells liberate a number of various other substances which include: the leukocytosis-promoting factor (L.P.F.), the thermo stabile leukocytosis factor, the leukopenic factors (leukopenin and the leukopenic factor), necrosin, pyrexin, leukotaxin, exudin and apparently also a diffusible growth-promoting factor, or factors concerned in repair.

Cytology: In the management of respiratory allergy and infection, the routine cytologic examination of the nasal, bronchial, salivary and other secretions for the presence of eosinophiles, neutrophiles and epithelial cells is of inestimable value. More recently Bryan[8] has emphasized the significance of changes in the nasal epithelial cells, exhibited by a definite pattern of degeneration, pathogenic of virus infections such as the common cold, herpes labialis and measles. Changes characteristic of allergy may also be noted. Exfoliative cytology, using the Papanicolaou technique, may be employed like also in the diagnosis of malignant lesions. The interpretation of these cytologic responses and changes should be the responsibility of the otolaryngologist and should not be relegated to the pathologist. In most instances immediate diagnosis is imperative, so rapid methods of staining must be employed, using the Hansel[4] or Wright stain, especially in the management of allergy and infections.

ANTIBIOTISM AND IMMUNITY.

Although the antimicrobial agents have been indispensable in the treatment of various infections, they have introduced new problems which concern chiefly[9] hypersensitivity reactions, malnutrition, monilial infections from alterations of local flora, resistant strains of organisms, abnormal mitoses and lowered antigenicity resulting in poor antibody stimulation and alteration of the usual clinical course of infections. The development of these factors has been the result apparently of the promiscuous and unnecessary widespread use of the antimicrobial agents. The changes in the clinical pictures are characterized by the masking or mildness of the symptoms out of proportion to the existing pathology. There is often a striking absence of the resolution stage of the infectious process with a resultant prolonged stage of healing. Frequent recurrence of respiratory infections because of apparent lack of immunity, is now a common observation.

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employing the proper therapeutic approach. In the first place, there is urgent need for effective respiratory vaccines which may be used alone or with antimicrobial agents. By purely clinical experimentation and observation extending over a period of about 10 years,[10] we have found that certain vaccines are very effective if the material has antigenic potency, and the optimum dose is administered at the proper time intervals. Raffel[11] has recently emphasized the importance of the proper selection of bacterial antigens if satisfactory immunity is to be produced. Our initial studies began with the use of Staphylococcus toxoid in the treatment of respiratory infections and external otitis (furunculosis). In many instances of acute respiratory infection, especially when the deep intradermal injection of 1/100 to 1/1,000 of a unit of Staphylococcus toxoid was given early or at the onset, marked attenuation or complete abortion of the infection occurred.

Although these striking results were not noted in a large percentage of cases, when they did occur they were dramatic, and they are worthy of further experimental and laboratory investigation. The effectiveness of Staphylococcus toxoid was apparently due to the optimum dosage and because the infection was caused by the Staphylococcus. In those instances in which other related organisms such as Pneumococci, Streptococci, H. influenza, etc., were the cause of the infection, immunity could result from the anamnestic response or reaction. Similar and even more consistent good results have been obtained in the treatment of external otitis with 1/100 unit doses at three day intervals. Swelling and pain may subside within four to six hours.

In the vaccine therapy of respiratory infections, further studies are being conducted with Streptococcus toxin, pneumococcus polysaccharides, specific and polyvalent virus mixtures, supported by pertinent laboratory procedures.

According to the observations of Komis,[12] antibiotics suspend the multiplication and the injurious action of microbes, but do not immunize the individual against the same infectious pathogenic microorganism. He states further that the lack of natural immunity and, especially, acquired immunity are the main and only reasons for the incomplete and unstable curative action of antibiotics today. The answer to the problem is dependent upon the concomitant use of vaccines. The respiratory vaccines in use today are prepared from killed organisms. They are toxic rather than antigenic in action, so satisfactory immunity is not produced by their use. Komis has developed a method of preparation of vaccines such as those from Staphylococci, Streptococci, etc., by an alcoholic fermentation process. He has demonstrated that they are highly antigenic and extremely immunizing, as indicated by biologic, experimental and clinical methods. No local or general reactions have been noted from their therapeutic use.

The most commonly encountered problem in otolaryngologic practice today is the patient with a low grade sinusitis, serous otitis or bronchitis, which has persisted for four to six weeks or longer, beyond the normal resolution stage of seven to ten days. It is evident, clinically, that this situation is caused by lack of immunity and low antigenicity of the infecting organisms, resulting from the widespread use of antimicrobial agents and the failure to balance therapy by the simultaneous use of vaccines with antigenic potency administered in the proper optimum dosage, and within the intervals of effectiveness.

The choice of the vaccine and the antibiotic should be based upon bacterial cultures and upon *in vitro* sensitivity tests. In these cases of subacute respiratory infections, vaccine therapy alone, or combined with the use of antibiotics, should be employed along with the mechanical removal of secretions from the sinuses and tracheobronchial tree. This can be adequately accomplished by the inhalation of Alevaire (Winthrop) in the form of an aerosol, as recently reported by Miller.[13] It decreases the viscosity and dilutes the secretions so that they may be removed more easily from the sinuses by suction, or from the bronchi by coughing. In the more persistent and severe cases, inhalations should be given once or twice daily; in average cases every two days. Marked improvement or complete recovery occurs in seven to ten days. For each treatment 8 to 10 cc. of Alevaire are aerosolized within 45 minutes to one hour. During the treatment the patient is instructed to cough up the secretion several times, and in some instances postural drainage is advisable. The apparatus employed consists of a DeVilbiss Compressor No. 501 with a No. 640 Nebulizer attachment, or the Aeromist pump with their Hi-flow Nebulizer.

In cases of tubal obstruction, Alevaire, ephedrine, antibiotics, etc., may be aerosolized and introduced into the Eustachian tubes very effectively by having the patient close the nostrils with the nasal tip attachment, then swallowing, with the aid of a mouthful of water if needed.

PRESENT STATUS OF TONSILS AND ADENOIDS.

In making a decision regarding the tonsil and/or adenoid operation, especially in children, on the basis of sore throat, frequent colds and bronchitis, allergy must always be considered as a factor. (Craft,[14] Clein[15]), Hansel and Chang,[16] found that about 12 per cent of the candidates in children had respiratory allergy. Since many of the respiratory and ear infections are now of a much milder type, and since they are more recurrent than formerly, on account of the lack of immunity, it is difficult in many instances to foretell whether the removal of the tonsils and adenoids will decrease the incidence of infections.

DRUG ALLERGY.

In the prescribing of drugs for the symptomatic relief of otolaryngologic conditions the possibilities of drug allergy,[9] idiosyncracy or intolerance always exist. Even therapeutic doses vary in different individuals. In allergic patients drug allergy may develop to aspirin, barbiturates, codeine, antihistamines, antibiotics, ACTH, etc., so it is important to recognize the fact that the patient may acquire a sensitivity to the drug administered to relieve certain symptoms.

Before administering antibiotics a careful history regarding previous untoward reactions must be taken. Serum disease reactions are more common from penicillin. Oral and gastrointestinal lesions or complications are more common with the broad spectrum antibiotics. It is also important to recognize urticaria, exfoliative dermatitis and purpura as sensitivity reactions. White blood and differential counts should be made for the possibility of granulopenia.

VASCULAR HEADACHES AND RELATED PHENOMENA.

One of the most common types of headache is the one described by Horton[17] as Histaminic Cephalalgia, in which the pain is usually located in the frontal, ocular and maxillary regions. The attack is often associated with lacrimation and stuffiness of the nose on the affected side. Pain in the antrum region and in the upper teeth may also be present. Since this syndrome simulates sinusitis, the patient usually consults the otolaryngologist for "sinus trouble". The attacks are not always typical of the Horton type, the onset may not be sudden, it may last for 12 to 24 hours or longer. In tension cases the onset may be more gradual, the attacks last longer and a tension factor is usually evident. Typical migraine, in our experience,[18] constitutes less than five per cent of the cases observed. Severe headaches of general type may be of allergic origin. In this type, edema of the brain occurs rather than simple vascular dilatation. Symptoms referred to the pharyngeal and laryngeal regions usually designated as "globus" may be of vascular origin.[19] Very few physicians are familiar with the management of headache problems, and since the otolaryngologist so frequently encounters these cases he should take the responsibility for their management.

In many instances vertigo and tinnitus are of vascular origin and may be managed by the same methods used in headache. Histamine, nicotinic acid, Dramamine, bonamine, etc., usually give good results, but in some cases no type of therapy may be of benefit. In severe Meniére's disease, destruction of the labyrinth may be indicated.

AUDIOLOGY.

During recent years, tremendous advances have been made in the field of audiology, especially in relation to the fenestration operation, chronic suppurative otitis media and deafness of all types. The fitting of hearing aids frequently requires patience, skill and adequate equipment. Audiology has, therefore, become a subspecialty of otology, and it may require more time than the busy otolaryngologist has to devote to it.

EFFECTS OF TOBACCO.

Many of the problems encountered in otolaryngology are directly or indirectly related to the use of tobacco, and the otolaryngologist has the opportunity to be of great service to his patients as well as to himself. The elimination of tobacco addiction should begin with the physician himself, if he is to be successful in the management of the patient.

It is well known that tobacco with its tars and other irritants, plays an important part in the cause of cancer of the lip, tongue, buccal mucosa, pharynx, larynx, esophagus and bronchi. The toxic effects of nicotine cause nasal stuffiness, loss of smell, decrease in mental efficiency, vertigo, deafness, blindness as well as many cardio-vascular lesions.

A primary sensitivity to tobacco should always be considered in allergic patients. No patient with nasal or bronchial allergy should smoke, and the parents of children with respiratory allergy should not smoke within the household.

Tobacco smoking may cause violent attacks of coughing which may result in vertigo, syncope or cerebral hemorrhage. The bronchial irritation from smoke, with coughing, aggravates bronchial asthma. The plugging of the swollen bronchi produces areas of atelectasis and eventually emphysema, with marked chronic dyspnea and pulmonary invalidism. The otolaryngologist should play the leading part among physicians in general in the widespread elimination of tobacco addiction.

GENERAL RELATED FACTORS.

Nutrition: A significant number of patients encountered in daily practice are underweight, anemic, undernourished and have poor dentition. These factors may play an important part in allergic states, susceptibility to infection, headaches, vertigo, possibly deafness, oral lesions, etc.

Spies[21] states that more than 70 per cent of patients observed by him, with clinical evidence of deficiency diseases, present lesions of the tongue, gums and buccal mucosa. To treat successfully patients with pellagra, scurvy, sprue, pernicious anemia, iron deficiency and related conditions, the changes of nutritional origin which affect the oral cavity should be recognized. Typical cases are rare, so the mild and atypical cases are the rule. Spies recommends dried Brewer's Yeast and liver extract for diseases caused by the deficiency of the Complex vitamins. In some instances larger amounts of specific vitamins such as vitamin C, B6, B12, folic acid and iron, or a complete vitamin supplement with minerals may be indicated.

Halpin[22] has pointed out recently that good nutrition is necessary for optimal resistance to infection and antibody production. Gastrointestinal upsets with vomiting and diarrhea cause rapid depletion of resistance. This not infrequently occurs with the administration of the broad-spectrum antibiotics, which also destroy the normal bacterial flora of the intestinal tract, with a resultant alteration or destruction of the biosynthesis of vitamins such as the B Complex and vitamin K. To combat this situation, these vitamins, as well as vitamin C, are now combined with the antibiotics. In the administration of ACTH and Cortisone compounds, nutrition is also important in the control of nitrogen balance, retention of sodium, loss of potassium, increased glycogenesis and glycosuria and the development of edema.

ENDOCRINES.

Proper endocrine balance must be a part of good nutrition. Thyroid deficiency may play a part in respiratory allergy, headaches, vertigo, etc. Estrogens, androgens, pituitary and adrenal hormones also play a part.

PSYCHOSOMATIC FACTORS.

Normal nutrition and endocrine balance may be directly or indirectly related to psychosomatic states. Allergic balance is often disturbed by such factors especially in nasal allergy and bronchial asthma.

As Davison has pointed out, many patients who consult the otolaryngologist have cancer and tuberculosis phobias.

They present symptoms for which there is no pathology or other explanation. In the psychologic management of all patients, assurance is important. The problem of diagnosis and the possible outcome of treatment should be explained to the patient so that he may know what to expect.

SUMMARY.

In the private practice of otolaryngology today, 80 per cent or more of the problems encountered require medical treatment. Such problems concern allergy, respiratory and other infections, the use of vaccines, antibiotics and other drugs, Alevaire aerosol, smoker's syndromes, headaches, vertigo, deafness, etc. Other supplementary factors require attention chiefly to nutrition, the endocrines, and psychosomatic states. Allergy, infections and headaches are the most important problems.

Nasal allergy and hay fever, with or without sinus involvement, must be distinguished from infection. The common cold is a frequent complication and may be diagnosed accurately only by cytologic examination of the secretion.

With the widespread use of antibiotics a situation has developed in which bacteriologic studies and *in vitro* tests are important in the selection of the proper antimicrobic agent. Since antibiotics do not produce immunity the resultant lack of it has increased the incidence of respiratory infections and has prolonged the duration of the illness. As a consequence, there is much need for vaccines with good antigenic properties and the establishment of optimum effective dosages.

Untoward reactions and complications following the use of antibiotics presents another problem which is not infrequently encountered, and which must be combined with adequate therapy and management.

As most headaches are referred to the region of the frontal, ethmoid and maxillary sinuses, the otolaryngologist is often the first to be consulted, because the patient believes he has "sinus trouble". Practically all these headaches are of vascular origin and not caused by sinus disease. Satisfactory results may be obtained in a large percentage of cases by the administration of histamine, nicotinic acid, symptomatic drugs and consideration of tension, psychosomatic, endocrine, nutritional and other related factors.

The smoking of tobacco may account for a number of common complaints and for malignant lesions encountered in the field of otolaryngology, such as stuffy nose, loss of smell and taste, postnasal drip, cough, dyspnea, precordial pain, vertigo, tinnitus, deafness, headaches, cancer of the lip, buccal mucosa, tongue, pharynx, larynx and bronchi.

Many patients need good medical management concerning nutrition, endocrine balance, psychosomatic management and above all, assurance as to the outcome of their complaints.

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BOOK REVIEW.

L'Education de L'Enfant Sourd par les parents, avant l'Ecole. Docteur Roger Maspetiol, Docteur Michel Soule, Docteur Josiane Guillamaut, Fernand Fourgon, Marcel Gautie. ESF, 17, Rue Viette, Paris 17e. Soft cover 191 pp.

This is a comprehensive volume on the education of the deaf child. It is intended to be of help to parents and teachers.

S. R. S.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

At the request of the Sixth International Congress of Otolaryngology, the American Express Company has arranged three post-Congress tours which will begin May 11, 1957, immediately after the close of the Congress. The tours will run concurrently and each will be accompanied by an American Express Tour Escort. All three will be in Philadelphia at the time of the Sixth International Congress of Bronchoesophagology, May 12 and 13, 1957. Tours 2 and 3 will be in St. Louis at the time of the International Congress of Audiology, May 14, 15 and 16, 1957, and Tour 2 will be in Chicago at the time of the International Voice Conference, May 20, 21 and 22, 1957. Other scientific events and a full social program are planned for the major cities visited. The itineraries for the tours are as follows:

Tour 1 (Eastern U. S.): 7 days. Cost $212.00 plus $4.10 Federal Transportation Tax. Cities to be visited include Baltimore, Philadelphia, Boston and New York.

Tour 2 (Middle West): 17 days. Cost $638.00 plus $15.98 Federal Transportation Tax. Cities to be visited: Baltimore, Philadelphia, St. Louis, Iowa City, Rochester (Minn.), Chicago, Detroit, Niagara Falls, Boston, New York.

Tour 3 (West Coast): 3 weeks. Cost $838.00 plus $30.47 Federal Transportation Tax. Cities to be visited: Baltimore, Philadelphia, St. Louis, Grand Canyon, Los Angeles, San Francisco, Chicago, Niagara Falls, New York.

The prices given include transportation, Pullman and hotel accommodations, meals and handling of baggage. Individuals from overseas who purchase tours abroad will be exempt from payment of the Federal Transportation Tax.

For detailed information and complete itineraries, communicate with the General Secretary, Sixth International Congress of Otolaryngology, 700 No. Michigan Ave., Chicago 11, Ill., U. S. A.

SCIENTIFIC PROGRAM OF THE AMERICAN OTOLOGICAL SOCIETY, INC.

The Seignory Club, Montebello, P. Q., Canada,

Friday, May 11, 1956

I. *Address of President—Introduction to a Symposium On the Utricle*..........William J. McNally, M.D.
II. *Microscopic Structure of the Utricle*..........Catherine A. Smith, Ph.D. (By invitation).
III. *The Question of Cortical Representation of the Labyrinth*..........Wilder Penfield, M.D. (By invitation).
IV. *The Importance of the Otolothic Organs in Man Based Upon a Specific Test for Utricular Function*..........Ashton Graybiel, M.D. (By invitation).
V. *(a) Postural Vertigo Due to Partial Destruction of the Vestibular Nerve;*
(b) Summary of the Symposium..........John R. Lindsay, M.D.
VI. *Autoradiographic Studies of the Intimate Structures of the Inner Ear*..........Leonard F. Belanger, M.D. (By invitation).
VII. *Analogy Between Cochlear Fluid Motion and Formation of Surf on Sloping Beaches* (Film presentation), Jurgen Tonndorf, M.D. (By invitation).

Saturday, May 12, 1956

I. *Address of the Guest of Honor—Hearing*..........Stacy R. Guild, Ph.D.
II. *Some Recent Experiments on the Neurophysiology of Hearing*..........Robert Galambos, M.D. (By invitation).
III. *The Part Played by Psycho-Galvanic Skin Resistance. Audiometry in the Testing of Hearing of Young Children*..........Page Statten, M.D. (By invitation), and D. E. S. Wishart, M.D.
IV. *Intravascular Agglutination of the Blood: A Factor in Certain Diseases and Disorders of the Ear*..........Edmund P. Fowler, M.D.
V. *The Structure of Bone and Cartilage*..........Robert A. Robinson, M.D. (By invitation).
VI. *Functional Repair of the Middle Ear in Chronic Otitis Media*..........Professor Dr. H. Wullstein, Wurzburg, Germany (By invitation).

SCIENTIFIC PROGRAM OF THE SEVENTY-SEVENTH ANNUAL MEETING, AMERICAN LARYNGOLOGICAL ASSOCIATION.

The Seignory Club, Montebello, P. Q., Canada,

May 13th and 14th, 1956

Sunday, May 13th

A. M.

Introduction Guest of Honor - - George M. Coates, M.D.

Remarks by the President Bernard J. McMahon, M.D.

I. *"Humidity as Problem in Air Conditioning"* Arthur W. Proetz, M.D. Discussor, Lawrence R. Boies, M.D.

II. *"Histamine Releasing Drugs"* - F. C. McIntosh, M.D. (By invitation). Discussor, Aubrey G. Rawlins, M.D.

III. *"Otolaryngologic Aspects of Hypometabolism"* Jerome A. Hilger, M.D Discussor, Francis W Davison, M.D.

2:00 P. M.

IV. *"Functional Diseases of the Nose"* C. Stewart Nash, M.D.

V. *"Unusual Virus Diseases in Otolaryngology"* Percy E. Ireland, M.D

VI. *"The Virus Theory of Nasal Polyp Etiology and Its Practical Applications"* Francis L. Weille, M.D. Discussor papers V. and VI., L W. Morrison, M.D.

VII. *"Management of Non-Malignant Growths in the Maxillary Sinus"* - O. E. Van Alyea, M.D. Discussor, Robert L Goodale, M.D.

VIII. *"Experimental Treatment of Recurrent Carcinoma of the Nasopharynx With Electrodesiccation, Radioactive Cobalt and X-ray Radiation"* .. ..Francis A. Sooy, M.D. Discussor, Harry P. Schenck, M.D.

Monday, May 14th

A. M.

IX. *"Ciliary Streaming Through the Larynx"* - Anderson C. Hilding, M.D. Discussor, George Edward Tremble, M.D.

X. *"Electrical Manifestations of Recurrent Laryngeal Nerve Function"*..................................John A. Murtagh, M.D. Discussor, C. J. Campbell, M.D. (By invitation).

XI. *"Submucosal Compartmentation of the Larynx"* (Colored Slides)..Joel J. Pressman, M.D. Discussor, Paul H. Holinger, M.D.

XII. *"Carcinoma of the Larynx"*..Fernand Montreuil, M.D. (By invitation). Discussor, Henry B. Orton, M.D.

2:00 P. M.

XIII. *"Benign Growths of the Vocal Cord; a Critical Analysis of Pre-operative Diagnosis Compared With Microscopic Diagnosis"*..................................Samuel Salinger, M.D. Discussor, Francis E. LeJeune, M.D.

XIV. *"Primary Malignant Tumors of the Uvula"*...........Fred Z. Havens, M.D., Richard C. Ye, M.D. (By invitation). Discussor, LeRoy A. Schall, M.D.

XV. *"Presentation of Two Laryngeal Cases"*.................John B. Erich, M.D.

SCIENTIFIC PROGRAM OF THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.,

Sheraton-Mt. Royal Hotel, Montreal, Canada,

May 15-16-17, 1956

Tuesday, May 15, 1956

(Mornings Only).

9:30

1. *Presidential Remarks*..................................Dean M. Lierle, M.D., Iowa City, Iowa.

Word of Welcome..........................Mr. Vincent Massey, Governor General of Canada.

9:40

Introduction of Guest of Honor..............Harold I. Lillie, M.D., Rochester, Minn.

Scientific Session

9:50

1. *Evolution of Surgical Technique in the Treatment of Carcinoma of the Larynx* Chevalier L. Jackson, M.D., Philadelphia, Pa. (Co-author: Charles M. Norris, M.D., Philadelphia, Pa.) No discussion.

10:10

2. *Developmental and Adult Anatomy of the Auditory Ossicles in Relation to the Operation for Mobilization of the Stapes in Otosclerotic Deafness*..............Barry J. Anson, Ph.D. (Med. Sc.), Professor of Anatomy, Northwestern University Medical School, Chicago, Illinois (By invitation). Theodore H. Bast, Ph.D. (Med. Sc.), Professor of Anatomy, University of Wisconsin Medical School, Madison, Wisconsin (By invitation). No discussion.

10:35

3. *Sick People in a Troubled World*........Howard A. Rusk, M.D., New York, N. Y., Associate Editor of the New York Times, Professor and Chairman of the Department of Rehabilitation and Physical Medicine of New York University's College of Medicine (By invitation). No discussion.

11:20

15 Minute Recess

11:35

4. *A Complication of the Stapes Mobilization Operation—Motion Picture*............................Howard P. House, M.D., Los Angeles, Calif. No discussion.

11:55

5. *Some Observations on Plasma Proteins in Relation to Allergy*................Bram Rose, M.D., Montreal, Canada, Associate Professor of Medicine, McGill University (By invitation). No discussion.

12:25

6. *Intracranial Complications of Otogenous Origin in Children Under Two Years of Age—The Role of Antibiotics*...............H. James Hara, M.D., Los Angeles, Calif.

12:45

Open Discussion.

Wednesday, May 16, 1956

9:40

7. *Structures of the Spiral Prominence and External Sulcus and Their Relation to the Organ of Corti*..................Merle Lawrence, Ph.D., Ann Arbor, Mich., Associate Professor of Physiological Acoustics, University of Michigan Medical School (By invitation). No discussion.

10:05

8. *One-Stage Repair of Hypopharyngeal Diverticulum—Motion Picture*......................Edgar S. Brintnall, M.D., Iowa City, Ia., Associate Professor of Surgery, State University of Iowa Medical School (By invitation). No discussion.

10:30

9. *Theory and Practice of Tympanoplasty*...................Professor H. Wullstein, Wurzburg, Germany, Universitats-Hals-Nasen-Ohrenklinik (By invitation). No discussion.

11:00

15 Minute Recess

11:15

10. *Automobile Fractures of the Larynx—Motion Picture* Authors, Paul H. Holinger, M.D., Chicago, Ill.; Kenneth C. Johnston, M.D., Chicago, Ill. (By invitation). Co-authors, Paul W. Greeley, M.D., Chicago, Ill. (By invitation), John W. Curtin, M.D., Chicago, Ill. (By invitation). No discussion.

11:30

11. *Tonsillectomy: Two Millennia of Hemorrhage and Controversy*.................................James T. King, M.D., Atlanta, Ga. No discussion.

11:50

12. *Corrective Surgery of the Nasal Tip*...............................John M. Converse, M.D., New York, N. Y. (By invitation).

12:10

Open Discussion.

Thursday, May 17, 1956

Scientific Session

9:30

13. *The Surgical Treatment of Carcinoma of the Anterior Commissure of the Larynx*..........F. Johnson Putney, M.D., Philadelphia, Pa.; Enrique A. Vicens, M.D., Philadelphia, Pa. (By invitation).

9:50

Open Discussion.

10:00

14. *Experiences With Streptomycin and Labyrinthectomy in the Treatment of Meniere's Disease*..........................Harold F. Schuknecht, M.D., Detroit, Mich. No discussion.

10:20

15. *Syphilis of the Larynx—in its Protean Manifestations—Motion Picture*..William A. Lell, M.D., Philadelphia, Pa.

10:40

Open Discussion.

10:50

10 Minute Recess

11:00

(1¾ Hour Limit)

16. *Symposium—The Operation for the Mobilization of the Stapes in Otosclerotic Deafness*........*Moderator*—Howard P. House, M.D., Los Angeles, Calif. *Participants*—John R. Lindsay, M.D., Chicago, Ill.; Victor Goodhill, M.D., Los Angeles, Cal.; Clair M. Kos., M.D., Iowa City, Ia.; George E. Shambaugh, Jr., M.D., Chicago, Ill.; Phillip E. Meltzer, M.D., Boston, Mass.; Edmund P. Fowler, Jr., M.D., New York, N. Y. *Remarks*—Samuel Rosen, M.D., New York, New York.

Open Discussion.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

PROGRAM 1956

Friday, May 4, 1956

6:30 P.M.—Cocktails

7:30 P.M.—Dinner—Dance

Cipango Club—3418 Gillespie

ANNOUNCEMENT.

The Mount Sinai Hospital, New York, in affiliation with Columbia University announces an intensive postgraduate course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty given by Irving B. Goldman, M.D., and staff, July 14, 1956, to July 28, 1956. Candidates should apply to Registrar for Postgraduate Medical Instruction, The Mount Sinai Hospital, 5th Avenue and 100th Street, New York 29, New York.

OTOLARYNGOLOGICAL POSTGRADUATE COURSE.

Saint Luke's Hospital and the Cleveland Otolaryngological Society will present a postgraduate course May 9, 1956, in Prentiss Auditorium, Saint Luke's Hospital of Cleveland. 11311 Shaker Boulevard, Cleveland 4, Ohio.

Guest speakers will be Alden H. Miller, M.D., Associate Clinical Professor of Otolaryngology, University of Southern California, Los Angeles, Cal., and Kenneth M. Day, M.D., Professor of Otolaryngology, University of Pittsburgh, Pittsburgh, Pa. Other participants in the program are H. C. Rosenberger, M.D., Fred W. Dixon, M.D., and William F. Hulse, M.D.

For further information write Paul M. Moore, Jr., M.D., Assistant Surgeon, Department of Otolaryngology, Saint Luke's Hospital, Cleveland, Ohio.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Wm. J. McNally, 1509 Sherbrooke St., West Montreal 25, Canada.
Vice-President: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Secretary-Treasurer: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Seignory Club, Montebello, P. Q., Canada, May, 1956.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Bernard J. McMahon, 8230 Forsyth Blvd., Clayton 24, Mo.
First Vice-President: Robert L. Goodale, 330 Dartmouth St., Boston, Mass.
Second Vice-President: Paul H. Holinger, 700 North Michigan Ave., Chicago 11, Ill.
Secretary: Harry P. Schenck, 326 South 19th St., Philadelphia 3, Pa.
Treasurer: Fred W. Nixon, 1027 Rose Building, Cleveland, Ohio.
Librarian, Historian and Editor: Edwin N. Broyles, 1100 North Charles St., Baltimore, Md.
Meeting: Seignory Club, Montebello, P. Q., Canada, May, 1956.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Dean M. Lierle, Iowa City, Iowa.
President-Elect: Dr. Percy Ireland, Toronto, Canada.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Mount Royal Hotel, Montreal, Canada, May, 1956.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: John R. Lindsay, M.D., Chicago, Ill.
Vice-Chairman: James W. McLaurin, M.D., Baton Rouge, La.
Secretary: Hugh A. Kuhn, M.D., Hammond, Ind.
Representative to Scientific Exhibit: Walter Heck, M.D., San Francisco, Calif.
Section Delegate: Gordon Harkness, M.D., Davenport, Iowa.
Alternate Delegate: Dean Lierle, M.D., Iowa City, Iowa.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION

President: Dr. Daniel S. Cunning, 115 East 65th St., New York 2, N. Y.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia, Pa.
Meeting: Sheraton Mount Royal Hotel, Montreal, Canada, May 15-16, 1956 (afternoons only).

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill., October, 1956.

THE AMERICAN RHINOLOGIC SOCIETY

President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. James Chessen, 1829 High St., Denver, Colo.
Annual Clinical Session: Illinois Masonic Hospital, Chicago, Illinois, October, 1956.
Annual Meeting: Palmer House, Chicago, Illinois, October, 1956.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. D. M. Lierle, University Hospital, Iowa City, Iowa.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Palmer House, Chicago, Ill., October, 1956.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Joseph Gilbert, 111 E. 61st St., New York, N. Y.
Vice-President: Dr. Kenneth Hinderer, 402 Medical Arts Bldg., Pittsburgh, Pa.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16, N. Y.
Treasurer: Dr. Armand L. Caron, 36 Pleasant St., Worchester, Mass.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. J. M. Tato, Azcuenaga 235, Buenos Aires, Argentina.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
General Secretary: Dr. C. E. Muñoz MacCormick, P. O. Box 9111, Santurce 29, Puerto Rico.
Meeting: Fifth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: April 8-12, 1956, San Juan, Puerto Rico.
President: Dr. J. H. Font, Medical Arts Bldg., San Juan, P. R.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis, Mo.
General Secretary: Dr. Paul Holinger, 700 No. Michigan Ave., Chicago (11), Ill.
Meeting: Statler Hotel, Washington, D. C., May 5-10, 1957.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. William J. Hitschler.
Vice-President: Dr. Chevalier L. Jackson.
Treasurer: Dr. John J. O'Keefe.
Secretary: Dr. Joseph P. Atkins.
Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. Thomas F. Furlong, Jr., Dr. William A. Lell, Dr. Harry P. Schenck, Dr. Benjamin H. Shuster, ex-officio.

BALTIMORE NOSE AND THROAT SOCIETY

Chairman: Dr. Walter E. Loch, 1039 No. Calvert St., Baltimore, Maryland.
Secretary-Treasurer: Dr. Theodore A. Schwartz.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

President: Paul H. Holinger, 700 No. Michigan, Chicago 11, Ill.
Vice-President: Dr. Jack Allan Weiss, 109 No. Wabash Ave., Chicago 3, Ill.
Secretary-Treasurer: Dr. Stanton A. Friedberg, 122 So. Michigan Ave., Chicago, Ill.
Meeting: First Monday of each Month, October through May.

OTOSCLEROSIS STUDY GROUP.

President Dr Gordon D Hoople 1100 East Genesee St, Syracuse, N Y
Secretary Dr Lawrence R Boies, University Hospital Minneapolis 14, Minn
Meeting Palmer House, Chicago, Ill, October, 1956

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President Dr G C Otrich, Belleville, Ill
President Elect Dr Phil R McGrath Peoria, Ill
Secretary Treasurer Dr Alfred G Schultz, Jacksonville, Ill

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President Dr Arthur S Bristow, Princeton, Mo
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Chairman Dr J L Levine
Vice Chairman Dr Russell Page
Secretary Dr James J McFarland
Treasurer Dr Edward M O'Brien
Meetings are held the second Tuesday of September, November, January, March and May, at 6 30 P M
Place Army and Navy Club, Washington D C

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman Dr Frederick A Holden, Medical Arts Building Baltimore 1 Maryland
Chairman Elect Dr Sherman B Forbes 706 Franklin Street Tampa 2, Florida
Vice Chairman Dr William J G Davis, 1632 K Street N W, Washington 6, D C
Secretary Dr G Slaughter Fitz Hugh 104 East Market Street, Charlottesville, Virginia

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President Dr Howard L Mitchell Lexington Va.
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Vice President Dr Edgar Childrey, Jr, Professional Building, Richmond Va
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Annual Meeting May 26 June 2 1956

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Dr James K Stewart, Wheeling W Va
Secretary Treasurer Dr Frederick C [illegible] [illegible] W Va

THE LOUISIANA MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President Dr W L Hughes, [illegible] [illegible], Jackson, Miss
Vice-President Dr Ralph H [illegible] [illegible] Line Ave, Shreveport, La
Secretary Dr Edley H Jones [illegible] [illegible] St, Vicksburg, Miss

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. J. C. Peele, Kinston Clinic, Kinston, N. C.

Vice-President: Dr. George E. Bradford, Winston-Salem, N. C.

Secretary-Treasurer: Dr. J. D. Stratton, 1012 Kings Drive, Charlotte 7, N. C.

Meeting: George Vanderbilt Hotel, Asheville, N. C., Sept. 16-19, 1956.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Norman Eaddy, Sumter, S. Car.

Vice-President: Dr. J. H. Gressette, Orangeburg, S. Car.

Secretary-Treasurer: Dr. Roderick Macdonald, 333 East Main St., Rock Hill, S. Car.

Meeting jointly with the North Carolina Eye, Ear, Nose and Throat Society, George Vanderbilt Hotel, Asheville, N. Car., Sept. 17-18-19, 1956.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Chas. C. Grace, 145 King St., St. Augustine, Fla.

President-Elect: Dr. Jos. W. Taylor, 706 Franklin St., Tampa, Fla.

Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

THE GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. A. R. Hollender, 605 Lincoln Road, Miami Beach, Fla.
Vice-President: Dr. Curtis D. Benton, Jr.

Secretary: Dr. James H. Mendel, Jr., 7241 Red Road, Miami, Fla.

Meeting: Quarterly, at Seven Seas Restaurant, February, May, October, and December.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Edwin G. Grafton.
Vice-President: Dr. L. A. Furchgott.
Secretary-Treasurer: Dr. Hal W. Maxwell.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Leland R. House, M.D.
Secretary-Treasurer: Sol Rome, M.D.

Chairman of Ophthalmology Section: Stephen J. Popovich, M.D.

Secretary of Ophthalmology Section: Richard Kratz, M.D.

Chairman of Otolaryngology Section: Harold Boyd, M.D.

Secretary of Otolaryngology Section: Howard G. Gottschalk, M.D.

Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles, California.

Time: 6:00 P.M., first Monday of each month from September to June inclusive—Otolaryngology Section. 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Lester T. Jones, M.D., 624 Medical Arts Bldg., Portland (5), Oregon.

Secretary-Treasurer: Dr. John F. Tolan, 3419 47th Ave., Seattle (5), Wash.

Meeting: April 15-19, 1956.

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

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Chairman: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.
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Otolaryngology: Dr. Russell M. Decker, 65 N. Madison Ave., Pasadena 1, Calif.
Ophthalmology: Dr. Warren A. Wilson, 1930 Wilshire Blvd., Los Angeles 57, Calif.

Mid-Winter Clinical Convention annually, the last two weeks in January at Los Angeles, Calif.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
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CANADIAN OTOLARYNGOLOGICAL SOCIETY
SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President: Dr. G. S. Gorrell, 110 Medical Arts Bldg., Calgary, Alta.
Secretary: Dr. G. Arnold Henry, 170 St. George St., Toronto, Ontario.
Meeting: June 6-7, 1956, Chateau Frontenac, Quebec, Canada.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Antonio da Costa Quinta, Lisbon, Portugal.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting:

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting: Lima, Peru, 1957.

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Secretario: Dr. S. A[illegible]
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Presidente: Dr. Alfo[illegible]n P.
Secretario: Dr. Felix [illegible]no.
Tesorero: Dr. Mario [illegible] A.

ASOCIA[illegible] OTORRINOLARINGOLOGIA Y BRO[illegible]AGOLOGIA DE GUATEMALA

Presidente: Dr. Jul[illegible] 15 Calle Oriente No. 5.
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Second Vice-President [illegible] José Luis Escamilla, 5a Calle P
No. 48.
Secretario-Tesorero. [illegible]race Polanco, 13 Calle Poniente N

SOCIEDAD DE OTO-RINO-LARINGOLOGIA. COLEGIO MEDICO DE EL SALVADOR, SAN SALVADO

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Secretary: Dr. Daniel Alfredo Alfaro.
Treasurer: Dr. Antonio Pineda M.

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Secretario General: Dr. Don Alfonso Vassallo de Mumbert.
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PORTUGUESE OTORHINOLARYNGOLICAL SOCIETY

President Dr Albert Luis de Mendonca
Secretary Dr Antonio da Costa Quinta Avenida de Liberdade 65 1 Lisbon

SOCIEDADE PORTUGUESA DE OTORRINOLARINGOLOGIA E DE BRONCO ESOFAGOLOGIA

Presidente Dr Alberto Luis de Mendonca
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1 ° Secretario Dr Antonio da Costa Quinta
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Tesoureiro Dr Jose Antonio de Campos Henriques
Vogais Dr Teofilo Esquivel
Dr Antonio Cancela de Amorim
Sede Avenida da Liberdade, 65 1 °, Lisboa

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Vocales Dr Osvaldo Suárez Dr Nondier Asis R Dr Jorge Bergallo Yofre

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Secretario Dr Jorge Perelló 319 Provenza Barcelona
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SOCIEDAD MEXICANA DE OTORRINOLARINGOLOGIA

Apartado Postal 21815
Mexico, D F

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President Elect Dr Miguel Arroyo Guijosa
Secretary Dr Luis Vaquero
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A STUDY OF THE ROLE OF CERTAIN FACTORS IN THE DEVELOPMENT OF SPEECH AFTER LARYNGECTOMY: 1. TYPE OF OPERATION; 2 SITE OF PSEUDOGLOTTIS; 3. COORDINATION OF SPEECH WITH RESPIRATION.[†]

Part 3: Coordination of Speech With Respiration.[†]

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Loss of the vocal function as a result of laryngectomy involves not only removal of the sound producing mechanism but also exclusion of the continued use of the trachea and lungs to supply air for audible speech. Except in the case of the laryngectomized speaker who employs the reed type of

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[†] This paper, presented in three parts, is based on the research associated with "A Study of the Role of Three Factors in the Development of Speech after Laryngectomy Type of Operation, Site of Pseudoglottis, and Coordination of Speech with Respiration" an unpublished Ph D. dissertation (Northwestern University, 1954) by Evelyn Y Robe Parts 1 and 2 appeared in the March and April, 1956, issues of The Laryngoscope

The research for the study was undertaken at the suggestion of Dr Chevalier L Jackson, Philadelphia, and was supported by a grant from the Illinois Division of the American Cancer Society.

Credit should be given to Mr William W. Waldrop, Director, Speech and Hearing Rehabilitation Service St Luke's Hospital, for his advice and assistance in this study.

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artificial larynx, the process of pulmonary respiration is separate from that of phonic respiration.

This changed relationship between breathing and speaking frequently presents discouraging difficulties to the individual who is learning to use a substitute voice. It also provides what is perhaps the greatest point of controversy with regard to theory and methods of teaching post-laryngectomy speech: *i.e.*, whether air is inspired through the tracheostomy to the lungs at the same time that air is drawn orally into the vicarious chamber, and whether air is expired from the lungs as the oral air is used in speaking.

The literature reveals a sustained interest in investigation of the problem and nearly every article which discusses teaching methods includes strong recommendations for synchrony or asynchrony of the two processes, as the case may be.

The results of a study by Stern in 1920 of the pulmonary and phonic respiration of a large number of laryngectomized subjects, "reinforced by a great number of cases examined by Schilling", are reported by Kallen as follows:

> *1.* Types of phonic respiration in laryngectomized persons can definitely be classified.
>
> *2.* Two main types of phonic respiration seem to predominate: In one, the process of breathing is normal; in the other, renewed respiration occurs with each word, often with each syllable. In this type, the interference with the new speech mechanism by ordinary vital pulmonary respiration seems to persist.
>
> *3.* A number of forms, both mixed and transitional, fall between the two main types.
>
> *4.* In a great number of cases, the pneumograph provides a clue to the location of the vicarious air chamber and the pseudoglottis. The lower these two structures lie, the more the thoracic element is likely to play a part in the new way of speaking. This follows if one or the other component of respiration serves as an aid in "ex-

> pression" of air necessary to activate the pseudoglottis.
>
> 5. The type of phonic respiration in the laryngectomized person may change with time and come to a certain stability only after many years. The change may be connected with the migration of the air chamber and with such factors as concern facilitation of neuronic pattern, etc.[8]

On the basis of his observation, Stern recommended avoidance of synchrony between pulmonary respiration and the learned process of air intake and use for speech. He believed that when the two occur together, not only is the vocal sound *likely to be obscured by friction sounds at the tracheostomy* as the air passes through, but coughing may also result. He urged special attention to this factor during the early stages of vocal therapy.[8]

A case of synchrony between respiration and speech in a laryngectomized person was described by Burger and Kaiser in 1925, and reported in Kallen's article. Their conclusion was based upon analysis of pneumograph recordings of respiratory movements at the nasal entrance and in the upper thoracic, gastric and abdominal regions.[8]

Kallen also referred to Froeschels, who described a patient in 1928 whose respiration and speech processes were synchronous and who was greatly aided in his efforts to obtain air for speech by the normal respiratory movement of the thorax and diaphragm. Graphic recording of the movements showed that the inspiratory and expiratory phases of speech corresponded with the movements of normal respiration during speech. When the patient was examined Roentgenologically, two phases of the speech act were observed. During the first, the diaphragm descended and at the same time the esophagus was filled with air. The second phase consisted of the rising of the diaphragm and expiration, while the esophageal walls contracted, expelling the air in speech.

Other observations regarding the action of the diaphragm during production of substitute voice were summarized by Kallen as follows:

> The general view is that the diaphragm plays a much greater part in the new speech mechanism than is supposed. In a certain sense the two reservoirs for air interfere functionally. Fluoroscopy shows peculiar jerklike movements of the diaphragm, which in selected cases obviously serve to compress the distal end of the esophagus or the gastric air bubble proper, thus aiding in the expulsion of air. That these movements have no connection with vital respiratory function is proved by the fact that they are seen to occur also when the tracheal cannula is shut off. It would follow that the diaphragm functions as an auxiliary in the new speech process, but otherwise than in normal speech.[8]

The literature reveals that the period from 1930-50 was marked by great interest in the development of programs for teaching post-laryngectomy speech. Nearly all such programs included recommendations regarding breathing and speech coordinations, although in most cases, no data were offered to support the belief that one method was superior to another.

Kallen, in 1934, recommended the reconditioning of phonic respiration as the patient's first task, even though it meant dissociating "the behavior patterns of a lifetime". The dissociation was necessary, he thought, because the old respiration habits would hinder production of the new voice and cause fatigue. Alternative methods to achieve the reconditioning were suggested: either the patient could be taught to hold the thorax in the position which follows inspiration and, while holding the position, to take in air for speech; or, the patient could take in air for speech after the lungs had been emptied of breath. He added that the latter method might be easier, but in either case it was necessary to continue to practice until conscious control of the process had been achieved.[8]

In 1936, Morrison and Fineman recommended that after the process of substitute voice had been explained, the next step was "to have the patient begin to practice the swallowing of air, always between breaths". He was to be instructed to swallow as much air as possible, without moving the chest wall. As an aid to the process, he might place a finger tip over the tracheal tube "to remind himself that he must not breathe in or out" while swallowing air.[14] This method of dissociation evidently corresponds to the second method advocated by Kallen as given above.

Levin, in 1940, reported the following data on the "characteristics of esophageal speech, studied and compared with those of normal speech in a phonetics laboratory by spirometry and other physiologic procedures:

> *1.* Thoracic action for each syllable and breathing movements for phrasing are the same as those used in normal speech.
>
> *2.* The normal person draws air into the esophagus with the mouth open; the laryngectomized person learns to close the mouth and use the compressive movements of swallowing to force the air into the esophagus quickly.
>
> *3.* Three or four syllables can be said with one intake of esophageal air, or roughly, about 1 cc. of air for each syllable is required.
>
> *4.* The pseudoglottis must open frequently and easily for intake of air and must contract when syllables are being formed.
>
> *5.* The movements of the sternum and epigastrium are more vigorous than in the normal person; therefore, in the beginning, students of esophageal speech tire easily, and become dizzy and have other transient symptoms.[10]

As an aid in overcoming difficulty in dissociating "reflex respiratory movements from the newer mechanism", Gatewood in 1944, advocated that patients be instructed to close the tracheal opening with a finger and then "attempt to insufflate air into the throat during the act of chest expansion". Because chest expansion has always been associated with inspiration, however, it would appear that Gatewood is really recommending association of the two processes rather than dissociation.[4]

In a discussion of a post-laryngectomy rehabilitation program in 1947, Howie also recommended "dissociation of respiration from aspiration of air into the gullet." Otherwise, the noise of air entering and leaving the trachea would mask the person's speech. Howie added that a patient using this type of speech can produce three or four syllables on one intake of air and probably uses 1 cc. of air for each syllable.[6]

Jackson and Jackson, in their program for "teaching a patient to develop articulate speech without a larynx," advocated the following instruction:

> The patient should be taught to inhale air into his esophagus by the expansion of his thorax in the ordinary breathing way, but at the moment of inspiration, the cannula must be obstructed with the finger. The air not being permitted to enter through the trachea will enter through the mouth or nose and be drawn into the esophagus.[7]

The method is based on the theory that when air is first inspirated, then swallowed, inspiration and deglutition are dissynchronized through being made to occur consecutively. One of the method's chief advantages is that "it forestalls the acquisition of the undesirable habit of air intake between short words or between syllables of longer words."[7]

Koepp-Baker advises the speech clinician to make a direct attack upon "the tendency to breathe through the tracheotomy tube during the esophageal voice production. This results in air turbulency at the orifice of the tube and obscures much of the speech." The physiological basis for such advice is as follows:

> In normal speech controlled expiration is an unconscious process. The cerebral cortex integrates the total synergy of respiration, valvulation at the larynx and the chewing, sucking movements in the mouth for normal speech production. When the total speech pattern is modified by loss of the larynx, and the esophagus and

> pharynx are used for phonation this totality must be fractionated and respiration must be inhibited.[9]

The French phonologist, Tarneaud, stresses the importance of impressing upon the laryngectomized patient at the beginning of the treatment that the customary lung breathing must not be resumed for producing the new voice. He recommends teaching the patient to take a breath, swallow, and then to breathe out quickly. Immediately after swallowing, the sound is to be produced.[11]

Holding the breath while belching air is described by Gardner as one of the most important techniques for laryngectomized speakers to learn. The reason for its importance, he says, is that it permits the speaker "to use the abdominal wall and chest muscles to exert pressure with the greatest efficiency. If air is breathed out while trying to belch, the decreasing volume of the chest cavity tends to lessen the tension and pressure for belching."[3]

Mason, in 1950, expressed the opinion that because "the walls of the esophagus move directly as a result of movements of the thoracic cage," air can be "inspired" into the esophagus for esophageal voice by inflating the chest as in ordinary inspiration. The air is returned by raising the intrathoracic pressure and simultaneously saying syllables and words. Deliberate variations in pressure divide the syllables from each other and are independent from respiration. Mason explains that this is to avoid the noise of air blowing through the tracheal opening. She also describes associated abdominal movements which may occur at the same time, and which were formerly thought to be responsible for the separation of syllables and words. Research by Marland in 1949, however, showed that they were merely accessory since, "during a sustained conversation such movements, as detected by a hand placed on the abdomen, are found to fade away, although the voice continues unaltered in power."[12]

The literature reveals that those investigators who advocate synchrony between pulmonary and phonic respiration are decidedly in the minority. To the list of its proponents, the names of Burger, Kaiser and Froeschels having been mentioned earlier; only Stetson and Moolenaar-Bijl are to be added.

Stetson employed apparatus specifically designed to make an objective and qualitative analysis of post-laryngectomy speech. The three forms chosen for study were: the buccal whisper, voice produced with an artificial larynx, and esophageal voice. His findings in regard to the last include the following:

> *1.* The chest action for each syllable and the breathing movements for phrasing are those of normal speech.
>
> *2.* Only a little air is taken into the upper part of the esophagus; from 2 to 5 cc.
>
> *3.* This intake is made frequently and rapidly with the mouth and velum closed; it often fuses with the movement of the consonant. Intake of air every few syllables is the one thing contrary to ordinary speech which the patient has to learn.[15]

As a result of his observations, Stetson offered an explanation as to why ordinary expiratory movements for normal speech are unsuited to esophageal speech:

> Intelligible speech cannot be made with an air supply exerting continuous pressure on the normal larynx or a substitute larynx, although that is a common assumption. The air pressure must fall to zero between the small groups of syllables, and often between syllables, and a separate pressure pulse must be made for each syllable. The one available mechanism for syllable production is the normal respiratory mechanism. Records of excellent esophageal speech show that the subject breathes just as in normal speech.[16]

Stetson concluded that a small supply of air frequently renewed could be controlled more efficiently by the original speech movements of the chest and that phrasing and accentuation would sound more natural as a result. His visual records showed that "the syllable grouping, phrasing, and accentuation are produced by just such expiratory movements as in normal speech; the only difference lies in the fact that the trachea is open and the movements are somewhat more vigorous to compensate."[16]

The practice of gulping a large amount of air for substitute voice was strongly opposed by Stetson. He believed that such an intake results in continuous pressure in the esophagus contrary to the situation in normal speech and makes the utterance "hasty, forced, belching in character."[16]

Moolenaar-Bijl, in 1951, confirms Stetson's findings. As the basis for her recommendation that the natural coordination of phonation and respiration should be maintained, she expressed the opinion that physiologically and psychologically it seems the most economical way.[13]

In the course of her investigations, Moolenaar-Bijl found that phonation always appeared to be coupled with expiration. Two types could be distinguished, according to whether the swallowing or sucking technique was used. When spirometrical curves were made of the speech of the patient who used the swallowing method, it was found that the mouth of the esophagus closed after phonation, which coincided with expiration, and remained closed until the next swallow, which occurred synchronously with inspiration. Observation of the esophageal curve when the patient was not speaking revealed absence of movement. This was confirmed by X-rays which showed no air in the esophagus while the patient was silent.

Analysis of spirometrical curves of the speech of a patient who used the sucking technique led Moolenaar-Bijl to conclude that a parallelism exists between the tracheal and esophageal air movements. In speech as well as in quiet respiration, the curves of the trachea and the esophagus travelled up and down together. When X-ray photographs were made of this patient, the esophagus was observed to be filled with air before and during speech. In the process of speaking, air was pushed out of the esophagus with pulsatile movements in synchrony with syllables. Air was replenished between two respirations with little sucking movements of the tongue or lips, which were sometimes identical with the articulation of initial plosives.[13]

The results of an experimental study by Anderson, in 1950, of some of the elements of esophageal speech proved to be inconclusive for either synchrony or asynchrony of the speech and respiration processes. A tape recorder and pneumopolygraphs were used to make simultaneous recordings at the mouth, thorax and abdomen. Analysis of the recordings revealed the following characteristics:

> Although great variation was noted from speaker to speaker, three relationships are apparent with reference to intake of air into the mouth and movements of the abdomen and thorax: *1.* Fixation of the respiratory movements of the abdomen and thorax takes place during the intake of air into the esophagus. *2.* Intake of air into the mouth is neither significantly synchronous nor asynchronous with inspiratory movements of the abdomen and thorax. *3.* Generally speaking, the abdomen and thorax exhibit synchrony of gross patterns if the record is inspected as a whole, but this synchrony does not exist if the tracings are compared for small detail.[1]

The foregoing review has shown that the majority of investigators are of the opinion that dissynchronization of breathing and speech in the laryngectomized speaker is desirable or necessary, while a minority believe that synchronization is preferable. Examples of both undoubtedly exist, and it is highly probable that some speakers shift from one to the other. It is also possible that some of those who recommend that the laryngectomized speaker learn to dissociate the two processes do not believe that the dissociation should be continued after the new speech is well-established.

The question of whether synchrony or asynchrony between breathing and phonation should be taught in post laryngectomy speech therapy is of fundamental importance to all those attempting to teach or to learn esophageal speech.

To study this relationship between phonic and pulmonary respiration, two complementary procedures were employed. *One used a graphic recording process; the other, motion picture photography.* Twenty-three laryngectomized persons who were classified as fair, good, or superior speakers were selected

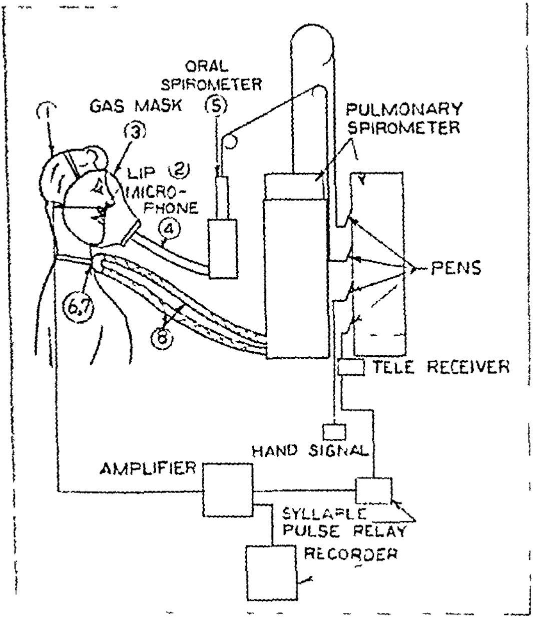

Fig 1 Diagram of apparatus used to record pulmonary and phonic respiration

as subjects for both parts of the procedure. The techniques in each part are reported separately and in enough detail to enable others to duplicate or extend the work if desired.

Part I: The first procedure involved the modification of equipment ordinarily used for studying the normal respirasometimes identical with the articulation of initial plosives.[13]

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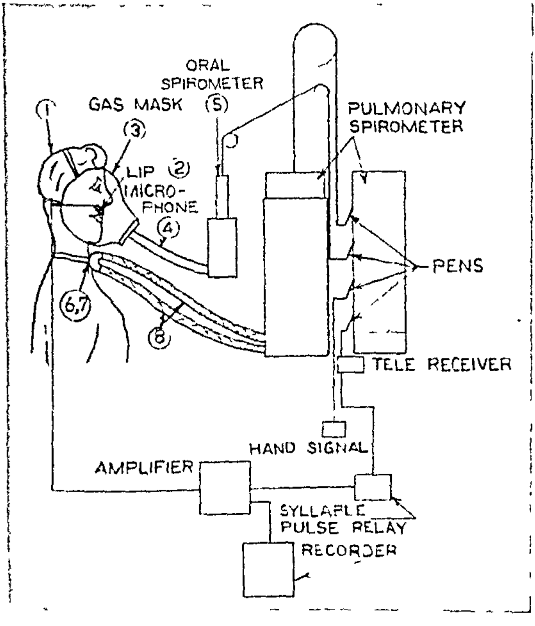


Fig 1 Diagram of apparatus used to record pulmonary and phonic respiration

as subjects for both parts of the procedure. The techniques in each part are reported separately and in enough detail to enable others to duplicate or extend the work if desired.

Part I: The first procedure involved the modification of equipment ordinarily used for studying the normal respiratory function. A description of the equipment follows, accompanied by a diagram (see Fig. 1), and photograph (see Fig. 2).

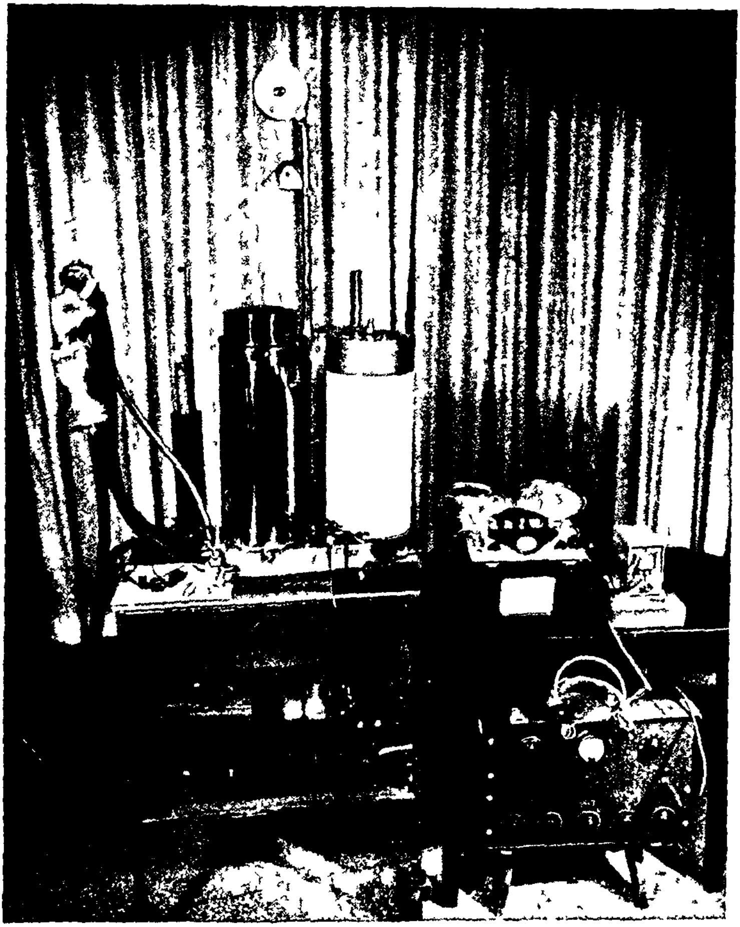


Fig 2 Photograph of apparatus shown diagrammatically in Fig 1

The subject (1) was seated next to the equipment. A lip microphone (2) was placed over the subject's upper lip and suspended from wires which looped over the ears. A civil defense-type gas mask (3), covering the entire face, was fitted on the subject. The mask was made airtight, with the addition of sealed foam rubber strips around the edges, and with the careful adjustment of the straps around the head. The cartridge had previously been removed from the front of the mask and replaced with a metal diaphragm and connection for $\frac{3}{8}$-inch rubber tubing (4), which led from the opening to the water-sealed oral spirometer (5). This was a specially built simple spirometer with a displacement of 1 cc. per mm. Its purpose was to record the volume of air the subject used in speaking.

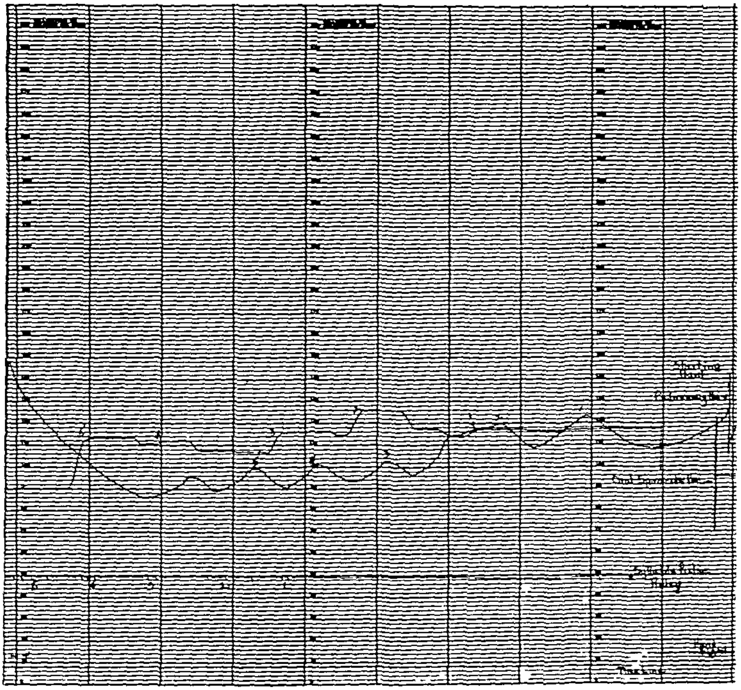
A laryngectomy or tracheotomy tube (6) of the correct size for the individual subject was placed in the tracheostomy, with a cushion of sealed foam rubber between the skin and outer rim of the tube, to insure an airtight connection. For the patient who did not use a tube, a special contact tube with sealed sponge rubber pad was placed over the tracheostomy. A metabolism mouthpiece (7) with the flat part removed was slipped over a rubber stopper on the laryngectomy, tracheotomy or contact tube, making it possible to connect a length of rubber tubing (8) from the tube to the large spirometer. This was a Benedict-Roth 9-liter spirometer with extra large size valves and soda lime absorber.

The customary speed of the paper drive mechanism was modified by the addition of a synchronous motor so that the paper moved at a speed of approximately 25 mm. per second.

As the drum revolved, four ink-writing pens recorded the data desired. The pen connected with the large spirometer recorded pulmonary respiration; the pen connected with the oral spirometer recorded the volume of air used in the production of speech; the third pen recorded the speech signal produced by the subject and transmitted through the following route: microphone, high-fidelity audio-amplifier, syllable pulse relay, and telegraph receiver; the fourth pen recorded a signal which was operated manually to indicate when words or phrases were given to the subject and when the subject spoke. The fourth pen was also attached to a timing circuit on the synchronous motor.

When the equipment was ready, the subject was brought into the room, and the purposes of the procedure in which he was about to participate were explained. The face mask, and tube connecting the tracheostomy with the large spirometer were fitted and adjusted and all connections tested for air leakage.

The recording of the speech and respiratory data for each subject consisted of four sections. During Section I, the subject was asked to breathe quietly without speaking. Only the pulmonary spirometer pen functioned during this period. For Section II, the subject was asked to count from one to five at his usual rate and volume (see Fig. 3). In Section III, the subject was asked to repeat five words from a standard "phonetically balanced" list,[2] while in Section IV, he was asked to repeat each of eight five-syllable phrases which were equated



Section II Counting.

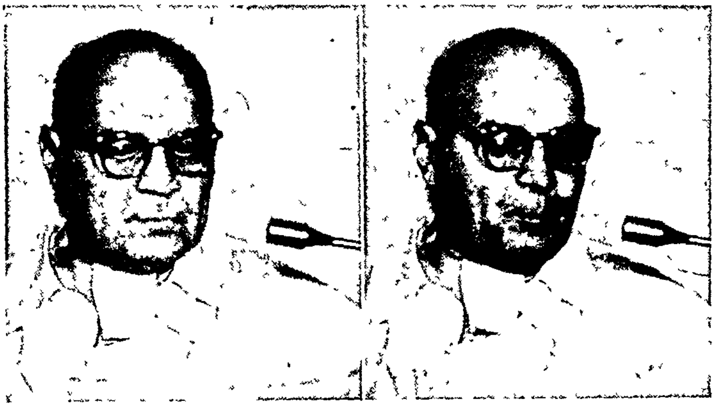
Fig. 3. Representative recording of speech and respiration data (Section II, Counting one to five).

as to time and intensity.[5] During the run, appropriate marks were put on the paper to identify the material. All sounds produced by the subject were recorded on magnetic tape. Time signals, at one-half second intervals, were also recorded on the paper, on a repeat run.

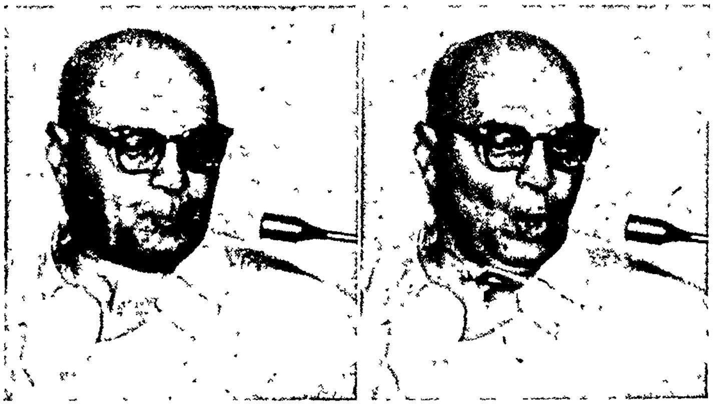
The procedure in the analysis of each completed record was as follows: *1.* A gross inspection was made of the four sections of the record. *2.* The volume of air taken in during the inspiration phase and the volume exhaled during the expiration phase of each cycle of pulmonary respiration were measured in cc. for each of the four sections. To determine these volumes, it was necessary to measure the number of millimeters for each phase of the cycle and to multiply the number by 20.73, which was the volume in cc. of each mm. of vertical movement of the pulmonary spirometer. *3.* The volume of air taken into the mouth for speech and the volume of air used in each speech unit were also measured in cc. This was done by computing the number of millimeters for each phase of the cycle. Each millimeter recorded by the oral spirometer pen represented the movement of 1 cc. of air in that system. *4.* Careful study and comparison of the oral and pulmonary curves were made to determine how many occurred *synchronously and how many did not. It should be noted that* limited space made it necessary to offset the pens so that comparable moments are not on any vertical line. A transparent glass plate with appropriate markings which showed simultaneous instants on the several curves were used to read the tracings.

Part II: The second procedure involved the use of motion picture photography and speech recordings. The same number of subjects (23) participated.

The following preparations were made for each subject: *1.* the tracheostomy and the area around it were uncovered so as to be clearly visible; *2.* a square of single thickness facial tissue slightly larger than the tracheostomy was secured along its top edge with Scotch tape, which was then attached to the neck just above the tracheostomy in such a way that the tissue would be moved readily by the respiratory air. This delicate curtain provided a visible and sensitive means for observing the air movement during inspiration and expiration; *3.* the subject was then requested to read the same words and sentences which had been used in Part I, followed by two short paragraphs. These paragraphs were constructed to include the sounds which were found by Anderson[1] to be the most difficult for the laryngectomized speaker to produce. A film and sound recording were made as the subject read. Color



1. Quiet Breathing.
2. Intake of Air.



3. Start of Phonation with Raising of Flap.
4 Phonation: Flap blown out.

Fig. 4. Prints made from individual frames of 16 mm. moving picture film giving visual evidence of relation between phonic and pulmonary respiration.

film was used throughout and all films were made at sound speed, *i e*, 24 frames per second (see Fig 4)

The visual and auditory data provided by the films and recordings were analyzed to determine the relation between pulmonary respiration and speaking for each subject Every film was studied frame by frame in a viewer for close observation of the correlation between the stages of lip movement during speech production and movement of the tracheostomy covering The film was then projected at sound speed as the tape recording of the subject's speech was played, in order to study the relation between the audible production of the sound and evidence of movement of the tracheostomy covering

RESULTS

Part I Analysis of the data regarding volume of pulmonary air revealed that considerable variation in range of volume from section to section was characteristic of the records of the 23 subjects Wide variation in pulmonary volume from speaker to speaker was also observed when individual records were studied and compared, furthermore, variation of pulmonary volume was generally found to be as great within the sections of an individual record as was the extent of variation in the group as a whole The nature of this variation made it impossible to conclude whether a relationship exists between speech fluency and the volume of pulmonary air used for respiration by the laryngectomized speaker

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Part II Analy

ings confirmed the results of the respiratory tracings in regard to the coordination of pulmonary and phonic respiration. The two occurred synchronously in all of the subjects.

The results of this section of the study support Stetson's observation that "records of excellent esophageal speech show that the subject breathes just as in normal speech."[16]

SUMMARY.

Twenty-three laryngectomized speakers served as subjects for respiratory tracings and moving picture photography, to provide data to determine whether pulmonary respiration is significantly synchronous or asynchronous with the oral inspiration and expiration of air for esophageal voice. Volume of air used in the inspiration and expiration phase respectively of every cycle of pulmonary and phonic respiration were also computed.

Positive synchrony between pulmonary and phonic respiration was demonstrated in each of the 23 subjects.

It is apparent from these studies that there is a wide variation in the volume of pulmonary air and oral air in different patients. There is no correlation between *1.* the volume of pulmonary air and the volume of oral air in any individual patient; *2.* between speech fluency and volume of pulmonary air; or *3.* between speech fluency and volume of oral air.

It was not possible to determine accurately from the sound recordings whether or not there was any relationship between loudness of the voice and volume of either pulmonary or oral air.

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HOARSENESS IN LARYNGEAL PATHOLOGY.

A REVIEW OF THE LITERATURE.

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INTRODUCTION.

Three pathologic conditions, among others, frequently result in the vocal condition known as hoarseness. These three are laryngeal carcinoma, paralysis and tuberculosis. It is the intent of this paper to present a review of the pertinent literature relating to the symptom in each of these etiologic categories. Recent years have produced little on hoarseness, and this writer intends to re-present the subject of hoarseness for serious consideration by all specialists.

Hoarseness is a symptom of laryngeal pathology. It may well be symptomatic of other conditions, but a basic tenet held by otolaryngologists and speech therapists is that this symptom is a danger signal of the larynx.[13] An extreme view of this is represented by Jackson and Jackson: "The chief symptom of laryngeal disease is hoarseness; it is absent only when the cords and the motor mechanism are entirely free from disease."[11]

The exact nature of hoarseness has been a matter of debate and discussion, not so much from the point of view of origin or therapy, but more from the problem of defining and describing it. The answers have centered around acoustical terminology, physiological, neurological, mechanical and psychological. In spite of what appear to be extreme difficulties inherent in solving this matter of definition and description, and because of the deliberate attempt here to focus attention on pathology, it is my intent to treat hoarseness as do medical pathologists and practitioners, and to utilize their definitions and descriptions.

Description. Jackson and Jackson[11] state that hoarseness is a general term applied to a rough quality of voice. Frank's[4] definition has gained acceptance because it is somewhat more

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specific: hoarseness is "any alteration in the speaking voice which results in a roughness or rasping character in the voice." Speech therapists and acousticians would add to these in detail, perhaps not in spirit. Every laryngologist has heard hoarseness in speech, and though he might not desire to define or describe it, it seems sure that he would certainly recognize it.

In demonstrating hoarseness, the laryngeal mechanism has failed partially or totally in one or more of its three major functions. Normal phonation requires that the vocal cords are capable of approximation, tension and vibration. Should any of these movements receive interference, hoarseness may result.[11]

Approximation may be disturbed by a growth between the cords, by feebleness, by paralysis, by interference with the movement of the cricoarytenoid joint, or by cicatricial concave cordal edges.

Tension may be disturbed by paralysis, feebleness, fixation of the joints, thickening of the cord, or by cicatrices of the concave cordal edge. As Jackson and Jackson define tension, it is an elastic recoil reinforced by muscular support of the cords; it is not a stretching like a violin string.[11]

Vibration may be disturbed by thickening of the cordal edges, by a growth acting as a damper, by feeble tension, or by incomplete approximation. Actually, approximation, tension and vibration may all be interfered with by the aggressive, usurpative action of the ventricular bands. "These bands can produce only hoarse phonation which in these cases is substituted for cordal phonation."[11]

Krieger notes that in the inflammatory type of hoarseness, approximation of the cords is only slightly interfered with, but the tension is very much impaired, varying with the severity of the condition present while the vibration varies directly with the tension.[12] In line with this same philosophy of complete investigation, Frank claims that a patient with hoarseness requires a thorough examination, laryngoscopy, indirect or direct when the mirror examination is not satisfactory; an X-ray of the chest, and occasionally of the neck; Wassermann; complete blood count and urinalysis. Biopsy should be performed when there is the slightest suspicion of a malignancy.

LARYNGEAL CARCINOMA.

It is evident that laryngeal carcinoma demands immediate consideration. Recent clinical experience has demonstrated that it is curable in a large proportion of cases *if diagnosis is made early.* On the other hand, in situations of late diagnosis, the trend is reversed to almost complete lack of cure. "Of primary importance is the recognition by the lay and the medical public that persistent or intermittent hoarseness in an adult means cancer, until it is proven otherwise by proper diagnostic means."[9]

Laryngeal carcinoma comprises about two per cent of all human malignant cancers. Like cancer of the mouth this disease occurs most often in middle and old age, and predominantly in males. The incidence of intrinsic and extrinsic cancer is about equal.

Specifically, as far as age and sex of affliction is concerned, it has been found in one statistical study that of 500 patients, 92 per cent were males, and that the average male age was 60, while the average female age was 40.[21] It was further noted that in cases of carcinoma of the larynx, with no treatment being received, 50 per cent of the sufferers are dead within one year, and that 95 per cent are dead in two years; yet, with the proper diagnosis and treatment, the five year level is as high as 82 per cent, and possibly higher today.[20]

Etiology. The pre-existence of a chronic local irritation which so often may be shown to play a part in the causation of malignant growths in the oral cavity is not readily demonstrable in the case of cancer of the larynx. Long felt to be etiologic factors in laryngeal cancer are excessive smoking with inhaling, voice strain and syphilis; yet, research has yet to demonstrate a significant etiologic role in most cases. As Martin states, "The disease usually appears to arise spontaneously."[16]

That growths in the laryngeal mechanism are the most common cause of hoarseness is hardly to be denied. Zinn states that neoplasms have been found in the largest number of cases; further, he reports that the symptom depends upon the involvement of the motor mechanism, on the location of the tumor, and whether or not it interferes with normal laryngeal movements.[26] These movements are, of course, those described earlier: approximation, tension and vibration.

Benign growths of the larynx, too, are responsible for this vocal condition; in fact, some authorities feel that there are many more benign than malignant lesions of the vocal cords, and, after the removal of such a benign tumor, one should expect a nearly normal voice in nearly all cases. Easing the situation somewhat is the fact that benign lesions of the vocal cords usually do not constitute a threat to life. It must be remembered, though, that they do cause disability manifested by hoarseness, and some benign lesions may be forerunners of malignant lesions.[7]

Symptoms. The fact that hoarseness does occur in laryngeal carcinoma is well known. Zinn reports that in 25 cases of total laryngectomy, 20 were hoarse preoperatively (89 per cent); that of another 178 cases, 100 were hoarse; and that of a group of 144 carcinoma victims, 134 (93 per cent) were hoarse.[26] Jackson and Jackson found that of 410 carcinoma cases, 389 (95 per cent) were hoarse.[9]

A growth situated anteriorly in the anterior commissure may so prevent approximation of the cords as to cause complete loss of voice (aphonia); on the other hand, a larger soft growth located below the cords may not interfere with adduction, and give rise only to the symptoms of local irritation. Paroxysmal hoarseness is often observed in such cases, especially after loud talking. A small tumor situated above the cords may give rise to very little trouble and cause no subjective symptoms. "In the great majority of cases, however, hoarseness is the first manifestation," state Lederer and Hollender.[14] Agreeing with this point, Jackson and Jackson note that the importance of location of the growth on the symptom depends upon whether or not the motor mechanism is involved. "By motor mechanism, we mean the cord, the arytenoid cartilage, the cricoarytenoid joint or the nerves or muscles concerned in their motility."[9]

It must be stated that the symptom under consideration, *hoarseness*, may not be present while the condition, *carcinoma of the larynx*, may well be. It is, however, with the symptom that this study is concerned; and it must be remembered that one of the earliest, most important and most common symptom is that of hoarseness. The absence of hoarseness in laryngeal cancer usually occurs when the growth is located above the glottis anteriorly. The symptoms for an advanced supraglottal carcinoma are frequently phonatory:: "The voice becomes more and more affected as the growth increases in size and eventually it becomes a wheezing, rasping, almost aphonic voice that is quite suggestive to those who have heard it often."[10]

Vocal cord cancer is evident in practically all cases first by hoarseness. The progress of the disease is usually slow, and the hoarseness may persist as the only symptom for as long as one to three years in untreated patients before the airway is sufficiently diminished to produce dyspnea. As the disease infiltrates deeply into the vocal cords, complete aphonia results. If, on the other hand, the lesions tend to fungate rather than infiltrate, husky phonation may be accompanied by gradually increasing dyspnea as the aperture of the glottis is narrowed.

Diagnosis. Inasmuch as hoarseness can be the symptom of a mortal illness, this fact and the observation of a new growth on the vocal cord should lead to a diagnosis that will either prove or disprove a cancerous condition.[8] Lederer and Hollender claim that "A conclusive diagnosis involves a careful survey not only of the larynx and its environment, but also of the body as a whole. A malignant tumor occasionally resembles chronic hypertrophic laryngitis, or a benign fibroma."[14]

Laryngeal carcinoma actually has three symptoms: persistent pain and discomfort on swallowing, the appearance of cervical metastasis in cancer of the extrinsic larynx, and hoarseness in intrinsic larynx (vocal cords) involvement.[16] Obviously, hoarseness, although insidious in growth, remains the most outstanding symptom; Jackson and Jackson place their hope for accurate diagnosis on this symptom alone, feeling that it is the one symptom for a number of months, perhaps a year.[9]

Because the vocal cords are the site of most cancers, it appears evident that motility in phonatory and respiratory activities plays an important role in the development of the tumor. It is not known definitely what causes the malignancy, as was stated previously, although Jackson and Jackson state, "The location of 85 per cent of cancers on the most abused part of the larynx, the middle third of the cord, renders it logical, even if not probable, that vocal abuse is one cause of cancer of the larynx."[4]

Summary. It appears, then, that cancer of the larynx seldom occurs without hoarseness. Hoarseness, especially of the persistent type, is obviously a danger signal; however, there can be a cancerous growth within the larynx without hoarseness. In the main, though, hoarseness is the first sign of cancer, and should be the signal to the speech therapist to refer the case to medical practitioners for complete laryngoscopic and other examination.

LARYNGEAL PARALYSIS.

Introduction: Neurological disorders of the larynx generally result in malfunctioning of the musculature controlling phonation. If by paralysis or paresis, these muscles are interfered with partially or completely, the symptom of hoarseness, as well as other vocal abnormalities familiar to the practicing speech therapist, may appear.

Etiology. In general, in paralysis of the larynx, the voice may be hoarse, and the patient may not be able to speak above a whisper, and there is special difficulty with vowel sounds. A similar change occurs in laryngitis and in tumors of the larynx. In a unilateral laryngeal palsy, or in recurrent paralysis, the voice is low-pitched and hoarse, but occasionally there is almost complete unilateral paralysis without appreciable effect on phonation, due to unimpaired adduction and to crossing of the midline by the normal cord. In bilateral abductor paralysis speech is moderately affected, but in bilateral total palsy, it is lost. "In paralysis of the c [illegible] is supplied by the superior laryngeal nerve, the voice is hoarse and deep and fatigues quickly. In adductor palsy, usually psychogenic, the loss of speech is out of proportion to the involvement."[2]

The description of paralysis of the larynx can be limited to two aspects: the opening and the closing of the cords. In this respect, Jackson and Jackson offer simple definitions. They term abductor paralysis as failure of the glottis to open from lack of impulses normally transmitted by the recurrent laryngeal nerve.[10] Similarly they describe adductor paralysis of the larynx as an impairment of closing movements due to impaired innervation.[11]

Friedman summarizes the general belief, briefly stating that in all progressive organic lesions of the centers and trunks of the motor laryngeal nerves, the fibers supplying the abductors of the vocal cords become involved much earlier than the adductors.[5] According to Sjoberg,[22] there is some significance in the bilaterally symmetrical innervation of the larynx. In supranuclear lesions, *i. e.*, above the nucleus ambiguus, paralysis of the larynx can occur only when the lesions are so great that double-sided interruption of the corticobulbar fibers takes place. The paralysis is then bilateral and of the spastic type. On the other hand, unilateral paresis of the vocal cord can never occur through supranuclear lesions.

Sjoberg describes the paralysis of the vocal cords as due to interruption of the peripheral conduction as a result of injury to the recurrent and vagus nerves in the neck or cavity of the thorax.[22] As an example of the neurological involvement of the larynx due to activities in surrounding areas, Froeschels points out that until recently more than 50 per cent of the cases showed paresis or palsies in the region of the recurrent nerve after operations on the struma; however, this number has been remarkably reduced by modern surgical technique and by research in dissection of the distribution of the recurrent laryngeal nerve.

Some 85 years ago William Marcet attributed another influence to the development of paralysis of the larynx, saying that paralysis or weakness of the laryngeal muscles causing loss of voice very frequently attends hysteria.[15] This view has numerous supporters today and must not be rejected by the speech therapist

Lederer has described neural pathologies in a convenient fashion

> Organic paralyses of the larynx fall into four clinical groups neuropathic encephalopathic myopathic and a combined form of myopathic and neuropathic types When the paralysis is neurogenic in form there is absence of local pathologic change The myopathic form is characterized by the presence of some local lesion such as swelling tumor or ulceration A great deal of evidence has been presented to support Semon s law (that the abductor muscles—cricoarytenoid— of the larynx show a greater proclivity to motor impairment than the adductors—thyroarytenoid lateral cricoarytenoid interarytenoid transverse and oblique) Some laryngologists however, believe that there are other mechanisms involved undoubtedly other nerves or muscles keep the vocal cord or cords adducted despite the destruction of the major nerve supply (recurrent laryngeal)

In discussing the nature of abductor paralysis, Berry and Eisenson report that the most common accident is the severing of the recurrent laryngeal nerve in thyroidectomy, as did Froeschels[6] They further state that less frequent is a paralysis of this nerve through pressure of a goiter, or aneurysm of the aorta The circuitous routing of this nerve accounts for the major difficulties Since this nerve furnishes the motor innervation to the posterior cricoarytenoid muscles of the larynx, any interruption of impulses will result in a failure of the glottis to open (abductor paralysis of the larynx)[1] Another supporting view of the importance of the recurrent laryngeal nerve is presented by DeJong, who states that the most common type of laryngeal palsy is the result of unilateral recurrent nerve lesion[7]

A simple but complete
of adductor palsy has b
that the cricothyroid mus
This muscle is responsibl
called median or para-m
current laryngeal nerve [illegible]
visual observation [illegible]
current laryngeal [illegible]
external branch [illegible]
marked abduct[illegible]
airway[19]

When the paralysis is confined to the adductors of the vocal cords, according to Friedman, it is almost invariably bilateral and is due to functional disturbances in the central nervous system. The organic lesions produce either respiratory difficulty alone, or both respiratory and phonatory difficulties. A partial paralysis of phonation has been observed in multiple sclerosis and bulbar paralysis. Lead poisoning has been known to produce paralysis of the abductors as well as unilateral complete recurrent palsy.[5]

Three stages have been described in recurrent paralysis: DeJong notes that the first manifestation is lessened abduction on the involved side, due to isolated involvement of the posterior cricoarytenoid; next, there is tension, of secondary contracture of the adductors: and finally there is complete paralysis with the cord in the cadaveric position The uninvolved cord crosses the midline.[2] Maxwell sees it somewhat differently in respect to the eventual position of the cords in this condition when he reports that the paralyzed vocal cords come eventually to lie in the midline as a result of recurrent nerve injury.[17]

Another neuropathology of the larynx is concerned with tensor, or thyroarytenoid paralysis. It is simply defined by Jackson and Jackson as failure of the tensor mechanism due to lack of motor impulses.[10] These authors state that as an isolated affection tensor paralysis properly so-called is rare; yet, as a result of total cadaveric paralysis, it is common.

Symptoms. In the case of abductor paralysis of the larynx where the glottis fails to open, Berry and Eisenson describe the result as a completely voiceless condition of the patient, and the folds are abducted into the cadaveric position. Gradually, as the cords approach the midline, he is able to phonate, but does so with air waste and husky quality to the sound. Breathing is stridorous. If the paralysis is limited to one cord, the normal cord will move back beyond the normal excursion in an effort to compensate for the immobile cord which remains in the midline; the total effect on the voice is practically the same.[1]

In general, DeJong agrees with this description, but uses the term "hoarseness" in describing the effect of the palsy. He has observed that in unilateral abductor palsy the involved cord lies close to the midline and cannot be abducted on inspiration. Too, in his opinion, the voice may become hoarse, but in general phonation and coughing are but little affected, since adduction is normal. In bilateral abductor palsy, both cords lie close to the midline and cannot be abducted. DeJong notes that the voice may be hoarse; but phonation is often only slightly affected, because both cords can still be adducted, and coughing is normal.[2]

Hoarseness plays a part in the description of bilateral abductor paralysis given by Jackson and Jackson, for they have noted that the outstanding characteristic symptoms of bilateral recurrent laryngeal paralysis are dyspnea and stridorous breathing; the voice may be little or not at all affected, but it will be noted that the phonation must be interrupted for a stridorous inspiration at frequent intervals. In complete paralysis with a cadaveric position of the cords, according to these authorities, there is a wheezing sound to the cough, and the voice is a very hoarse stage whisper without true phonation.[10]

Strother, too, notes that hoarseness is not a symptom of this type of paralysis. By his description, when bilateral paralysis follows the more common course, there is an initial aphonia during which the cords are abducted in the cadaveric position. Gradually, the voice is recovered, but as the voice returns, due to changes of the cords toward the midline position, the glottis becomes narrower and respiration becomes increasingly difficult. He notes, however, that bilateral paralysis cases present themselves to the speech therapist much less frequently than do cases of unilateral paralysis.[24]

Jackson and Jackson agree with Strother that unilateral abductor paralysis is much more common than the bilateral variety. In addition, they find the lost motion occurs more frequently on the left side than the right and that unilateral paralysis is usually peripheral, due to lesions in the neck or the upper part of the thorax;[10] and with one cord paralyzed the patient may phonate perfectly but it will usually be discovered that there was a period when he was hoarse.[10]

It seems that with abductor paralysis, the symptom of hoarseness has not been listed as a common one; yet, there are definite changes in the ability to phonate normally, changes which the speech therapist should recognize.

Hoarseness, however, has definitely been attributed to conditions of adductor paralysis of the larynx. The chief symptom in unilateral adductor paralysis, according to Jackson and Jackson, is hoarseness; a wheezy husky voice accompanied by air waste on attempted phonation.[11]

DeJong describes adductor palsy differently, noting that the cords are not adducted in phonation, and the voice is either lost or cannot be raised above a whisper. He notes that the difficulty is almost always bilateral and is generally of psychogenic origin. Unilateral adductor paralysis is occasionally seen in trauma and in peripheral lesions; there is paralysis of one lateral cricoarytenoid with hoarseness and also impairment in coughing.[2] In the case of recurrent paralysis, DeJong summarizes by stating that "the voice may be coarse and husky, and there is loss of the ability to sing."[2]

In a total unilateral palsy, DeJong notes that both adduction and abduction are affected, and that the involved cord lies in the cadaveric position, motionless in misabduction. The voice is low-pitched and hoarse, and there is difficulty in coughing, but phonation may not be seriously affected since the normal cord may cross the midline.[2]

As far as the effect of tensor paralysis, Jackson and Jackson state that the voice is weak and husky with air waste; a little conversational use causes the patient to say his "voice is tired," or his "throat is tired."[10] DeJong describes a slightly different manifestation, observing that in bilateral thyroarytenoid paralysis the glottis has an oval instead of a linear appearance during phonation; the voice is hoarse, but there is neither dyspnea nor stridor. In paralysis of the arytenoid the glottis is closed only anteriorly during phonation, according to DeJong, and inspiration is normal.[2]

Phonation as well as respiration are disturbed in progressive bulbar palsy. In the later stages of the disease, the voice becomes monotonous and feeble. Hoarseness may supervene and go on to complete aphonia. The cough is weak and hollow. Laryngoscopic examination in the beginning of bulbar palsies may show relatively little, according to Friedman, and later on, paresis of the adductors appears with insufficient closure of the vocal cords so that phonation is difficult.[5]

Summary. In unilateral paralysis, a speech therapist's cooperation is of great value to aid the patient in obtaining maximum results during the time that the cord, which is healthy, crosses the midline as an attempt of the physiologic sensibility of the larynx to compensate for the immobile cord. In recent years, progress has been made by external surgical means to relieve the patient of respiratory embarrassment in cases of bilateral paralysis, according to Lederer. Such procedures immobilize and draw aside an arytenoid, thereby permitting a small but functional airway without influencing the phonatory responses too adversely.[17]

It appears, then, that in instances of abductor paralysis, phonation is not always affected; thus the disorder under study, *hoarseness*, may not be attributed to faulty abduction of the vocal cords; however, the opposite aspect, that of adduction, does seem to present vocal problems, including hoarseness, when its function is disturbed (paralyzed). This is especially true in cases of recurrent nerve paralysis. One additional neurological malfunction concerns the total unilateral paralysis, affecting both adduction and abduction, with a resulting hoarseness in the syndrome.

LARYNGEAL TUBERCULOSIS.

Introduction. Although it is not the most common disease entity resulting in or causing hoarseness, large numbers of individuals infected with pulmonary tuberculosis show symptoms of hoarseness, indicating a spread of the primary disease to the larynx.

By definition, tuberculosis of the larynx is a lesion therein due to *Bacillus tuberculosis.*[10] It is an infectious and contagious disease of the larynx and is secondary to pulmonary tuberculosis. It is very rarely primary and is usually chronic from the onset. It is characterized by infiltration, formation of tubercles, granulomata, and ulcers in the walls of the larynx, followed later in some patients by edema, perichondritis, chondritis, or even necrosis.[23]

Thomson, writing in England in 1924, stated that there was no specific disease of the larynx so common as tuberculosis.[25] Notwithstanding the differences in geography, economics, and time, current American medical experts claim that tuberculosis is still one of the commonest of laryngeal lesions, excluding acute and chronic laryngitis.[10] It is more frequently met with in individuals between the ages of 20 and 45. It attacks both sexes, but is more common in men than women to the ratio of 3:2. From 3 to 25 per cent of the patients with pulmonary tuberculosis manifest laryngeal tuberculosis.[14,23]

Spencer[23] quotes Dworetzky[3] as having found hoarseness in 82.6 per cent of one of his series of early cases. In his observations of this disease, Thomson came to similar conclusions to those of Ormerod when he found that in 477 patients who had a lesion of the larynx, the most common complaint was some alteration of voice. He noted that change of voice may come so insidiously and cause so little general inconvenience that a patient and his friends may never have observed that a previously clear and musical voice had become toneless, rough, harsh, or even hoarse.[25]

Etiology. The involvement of laryngeal tuberculosis is a progressive process, according to Thomson,[25] with the posterior commissure (arytenoids, interarytenoid region, and vocal processes) most commonly affected. Following this, the vocal cords become involved. Next in order comes the anterior commissure. Last is the epiglottis in the progressive nature of the disease.

It seems clear that the somewhat complicated mechanism of approximation and vibration, as well as tension, of the vocal cords can be deranged in many ways, any one of which would result in some degree of hoarseness. The approximation of the cords can be interfered with by irregularity in the edges of the vocal cords themselves, either nodular or edematous, or by the interposition of hypertrophic tissue from the interarytenoid space between the vocal processes of the arytenoids. Cord approximation can also be affected adversely by interference with the approximation or rotation of the arytenoid cartilages. This can be caused by lesions which interfere with the fibers of the recurrent laryngeal nerve. Such lesions occur as a fibrosis at the apex of the lung, pressure from enlarged glands, or infiltration at the penetration of the muscle by the nerve.

Rotation of the arytenoid cartilages can be disrupted by any of the above conditions as a result of laryngeal tuberculosis, as well as by tuberculosis infiltration in the region of the cricoarytenoid joint. This can occur with or without implication of the joint or the cartilage, and can affect the rotation of the arytenoids in such a way as to interfere with approximation or vibration.

The vibration of the cords may be disturbed in a number of ways. Paralysis of the internal tensor muscles by lesions affecting the recurrent nerve, atony of these muscles by tuberculous debilitation of the patient, or infiltration of the muscle by tubercles have the effect of abolishing the tone of the vocal cord, thus affecting the tension and the vibration activities. The cord remains bowed on attempting to phonate and the voice is a very weak sound, particularly when the condition is bilateral. Infiltration of the vocal cord, with or without edema, prevents proper vibration as well as interfering with approximation.

The role of the ventricular bands is no small one in cases of laryngeal tuberculosis. Swelling of these bands, protrusion of swollen and infected membranes from the mouths of the ventricle of Morgagni and subglottic infiltration have the effect of dampers of the vocal cords and again prevent proper vibration.

In brief, then, hoarseness is produced by any lesion, however small, of the vocal cords, and by any but the very smallest of lesions in the ventricular bands or arytenoids, according to Ormerod.[18] He does state, however, that lesions restricted to the epiglottis or even to the aryepiglottic fold do not produce hoarseness.

Symptoms. Because it is secondary to the pulmonary condition, laryngeal tuberculosis is late in developing, notes Zinn,[26] and exhibits varied symptoms. Painful swallowing exists even in the absence of ulceration. Lederer and Hollender note that difficulty in swallowing is common among those patients in the presence of marked ulceration and infiltration, especially of the epiglottis or aryepiglottic folds. Change in the voice is at first absent; however, hoarseness may follow a paroxysm of coughing or an effort at speaking. Alteration of the voice, say these authors, depends upon the degree of involvement of the phonatory apparatus.[14]

Thus, authorities are fairly well agreed that the symptoms of tuberculosis are, in the main, threefold: hoarseness, pain and dyspnea, occurring in the order of frequency. Ormerod[18] reports that hoarseness was noted 290 times in 300 cases in which the symptoms and the data had been carefully analyzed. He states that in the slighter degrees the variation is very noticeable. A patient who has an obviously husky voice one day may have a normal voice the next, and hoarse again two or three days later. Some individuals with this disease entity report they are hoarse on waking and improve during the day; others have the opposite experience, waking with a good voice which apparently tires during the day.

According to this same writer, Ormerod, the hoarseness of tuberculosis of the larynx is never raucous, as in a benign or malignant tumor of the cord. The voice may be bitonal or somewhat rough, but there is always a sensation of moistness about it, and as the hoarseness increases the impression is made of collected mucous in the larynx. This authority states that the voice weakens, and eventually there is nothing left but a moist whisper.

Spencer[23] agrees generally in the progressive nature of the symptoms. He found that the subjective symptoms often begin with slight hoarseness. This could be present only in the mornings after the patient arises, or after a change in position if the patient is kept in bed, or after a paroxysm of coughing.

Jackson and Jackson stress the fact that voice is weak, not just impaired in quality—often weak out of all proportion to the lesion visible in the mirror.[10] According to them, this is due to a myasthenia affecting the thyroarytenoid muscle, which is usually the first muscle to show the weakening that later affects all muscles of tuberculosis patients. This muscular weakness may produce hoarseness, even aphonia, before there is any tuberculosis lesion in the larynx. Usually, state Jackson and Jackson, the voice deteriorates until it becomes a rough whisper, as though somewhat forced. In the earlier stages the cords may approximate very well and their edges may look thin enough to vibrate, but they seem to be inefficient in the production of sound.

If the patient is not kept silent, talking often precedes the hoarseness, according to Spencer. Then, later in the disease, the hoarseness becomes constant and soon results in aphonia. Jackson and Jackson, too, strongly support the efficacy of a silence regimen. They seem to believe that the prognosis in the case of a patient who will not and cannot stop talking is bad; it is next to impossible for a patient who talks all the time to get well of a laryngeal tuberculosis. These authors also state that the same may be said for those patients who indulge in alcohol and tobacco.

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THE LOW COLLAR INCISION FOR WIDEFIELD LARYNGECTOMY.*

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INTRODUCTION.

Since Kocher[1] first popularized the low collar incision, it has been used by surgeons throughout the world for thyroid surgery as well as other surgical conditions in the neck. This incision has long been recognized as the one which will give satisfactory cosmetic results and which will allow adequate exposure of the surgical field.

The low collar incision was used by Col. Wendel Weller and the author at Letterman General Hospital in 1948 for widefield laryngectomy in a patient with squamous cell carcinoma of the larynx. Since that time the author has used this incision successfully in patients who require widefield laryngectomy and in those who require primary block dissection combined with widefield laryngectomy. When primary block dissection is combined with widefield laryngectomy, an extension of one end of the incision is carried upward over the sternocleidomastoid muscle to the mastoid process. In reality, such an upward extension of one end of the incision allows satisfactory exposure of the surgical field, and is J-shaped on the left side of the neck and reversed on the right side, as contrasted with the Glück-Soerensen incision which is U-shaped.

It is of further interest to point out that some years ago Babcock[2] used a high collar incision for laryngectomy, excising a lower round piece of skin and subcutaneous tissue for creation of the tracheostomy.

It is the purpose of this paper to record our experiences with the use of the low collar incision for widefield laryngectomy in patients with squamous cell carcinoma of the larynx who have been treated during the past eight years.

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ADVANTAGES.

1. Exposure of the surgical field is adequate. The upper flap can be dissected well above the hyoid bone, and the lower flap can be dissected to the suprasternal notch. Such wide exposure allows removal of the hyoid bone above, adequate handling of the strap muscles and thyroid gland lobes and isthmus, and removal of as many tracheal rings as necessary with the specimen. Once the larynx is removed and the closure of the pharynx and esophagus is accomplished, suturing of the flaps and creation of the tracheal stoma are readily done.

2. Cosmetically the low collar incision has the advantage of being placed below the collar line of the average male so that the scar and stoma are covered by the shirt collar. In the female, an appropriate necklace can be worn to cover this area.

3. For the most part the tracheal stoma which is created is round and symmetrical. In over 97 per cent of the cases, no cannula of any type need be worn by the patient postoperatively.

4. As pointed out above, primary block dissection can be done simultaneously with widefield laryngectomy by the upward extension of one end of the incision. Further, secondary block dissection can be done, using the conventional T-incision without jeopardizing the blood supply of the original flaps.

5. If a sinus tract or fistulous opening develops postoperatively in the area of the incision, drainage in most instances is toward the supraclavicular spaces (ends of the incision) rather than toward the tracheal stoma area.

6. The development of esophageal voice by the patient proceeds in the normal manner.

7. It will be found that the fascial layers beneath the platysma muscles are relatively bloodless during the creation of the flaps.

DISADVANTAGES.

1. The creation of a relatively large upper flap and the smaller lower flap is time consuming.

2 A higher percentage of postoperative sinus and fistulous tract formation with neck suppuration will develop in patients when the low collar incision is used, if one does not pay particular attention to meticulous postoperative treatment (as will be pointed out later)

3 This incision is not suitable for all patients who require widefield laryngectomy Patients with gross X-ray changes of the skin such as atrophy, loss of blood supply and telangiectasia following irradiation for carcinoma of the larynx should not have a widefield laryngectomy through a low collar incision since the flaps may slough In this instance a T-incision is recommended Experience has shown that patients who have recently been irradiated usually do not show skin changes which would contraindicate the use of the low collar incision

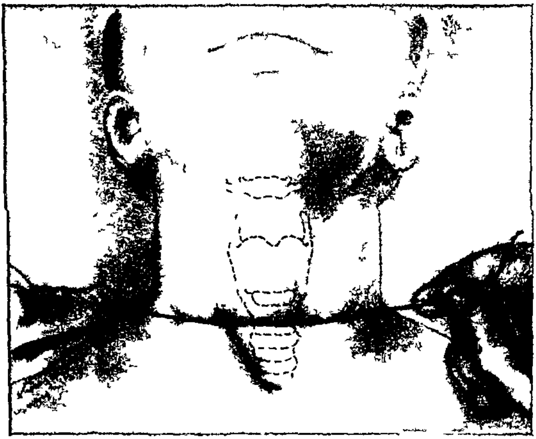

Fig 1 Low collar incision is being outlined by suture material

TECHNIQUE.

In performing widefield laryngectomy, the low collar incision is outlined by suture material between the lower border of the cricoid cartilage and the first tracheal ring, as shown (see Fig. 1). In most cases this approximates a distance of two finger breadths above the suprasternal notch. By thus marking the skin, the incision can be made symmetrical in relation to the surface contours of the neck. The surgeon can vary the outline of the incision as he desires in relation to the constant anatomical landmarks, namely, the cricoid cartilage and the first tracheal ring.

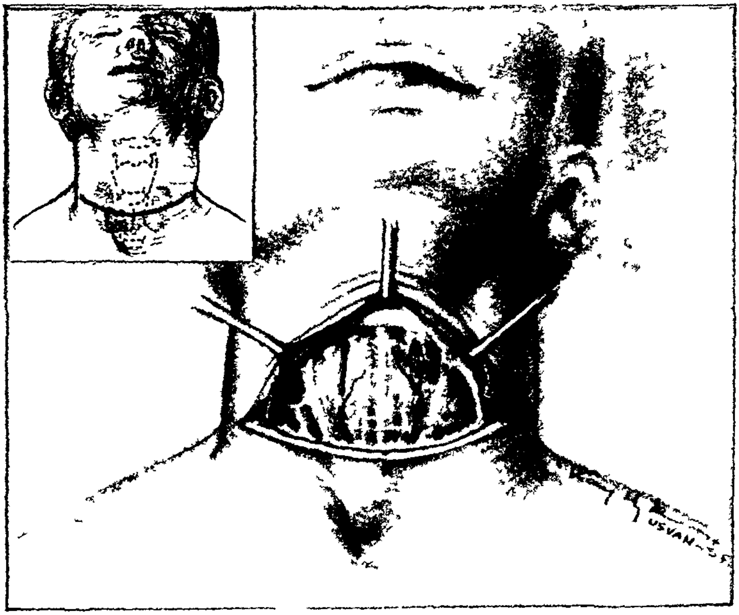

Fig 2 Insert shows vertical midline marking plus low collar incision outline The upper flap is being created

After the skin has been marked with suture material, a small vertical mark is made in the skin directly over the midline of the neck so that this mark can be used at a later stage in the operation to help approximate the upper and lower skin flaps to the severed trachea in both a dorsal and ventral position, respectively (see Fig. 2). The incision is carried through the platysma muscles, which are then lifted with the skin flaps thus exposing the superficial layer of the deep cervical fascia and venous channels (see Fig. 2).

The upper and lower flaps are completed, as illustrated in Fig. 3, with the cervical fascia, venous channels and muscles in view. The suprahyoid muscles and their fascia are exposed, and this allows complete removal of the whole hyoid bone with the larynx. Laterally, the bellies of the sternocleidomastoid muscles are exposed The inferior flap is elevated to the suprasternal notch, assuring adequate mobilization for closure. Complete mobilization of the lower flap aids closure, particularly in those patients with enlarged thyroid glands and in those cases where several rings of the trachea have been removed with the specimen.

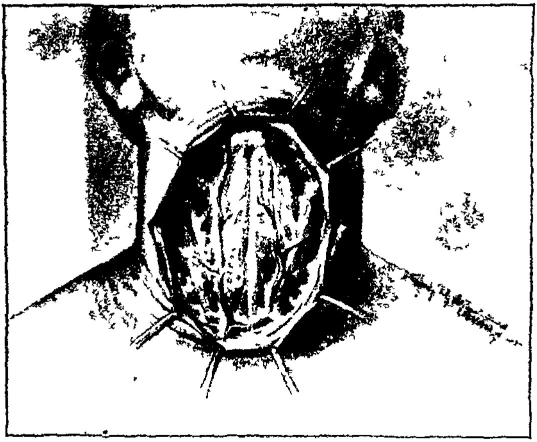

Fig 3 Upper and lower flap created showing adequate exposure of the surgical field

The widefield laryngectomy from this point on proceeds [illegible] a step-by-step manner and Fig. 4 shows the neck after [illegible] larynx and adjacent structures have been removed; in fact, closure of the pharynx and peripharyngeal muscles has been completed in a T-shaped manner. The severed strap muscles have retracted beneath the sternocleidomastoid muscle bellies and for that reason are not illustrated. The cut isthmus of the thyroid gland has pulled upward permitting the severed trachea to assume a more ventral position. Note that the rubber drains are placed high in the gutters of the wound, and are positioned laterally throughout. By placing the drains thus, the vulnerable T-closure area of the pharynx is allowed to be in apposition to the under surface of the flap from the time it is placed for closure.

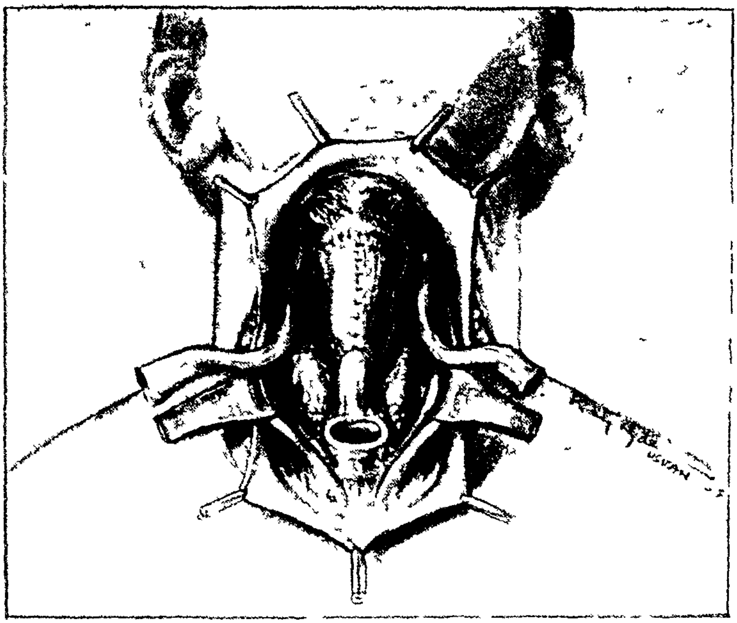

Fig 4 The larynx and adjacent structures have been removed and closure of the pharynx completed

After hemostasis is secured, and the drains have been placed, closure of the wound is completed. The formerly marked area of the midline of the upper flap is sutured to the dorsal midline of the trachea (see Fig. 5, trachealis muscle area). Even though an intratracheal breathing tube is in place, suturing is done about the tracheal stump. Skin is approximated to the tracheal mucosa at all times; and, laterally, tracheal cartilage is included in some of the individual interrupted sutures. These sutures are so placed that each includes a generous amount of upper flap skin so that there is an actual puckering of the skin about the tracheal stump. In healing this allows equal tension and pull of scar tissue about the stoma. The upper flap is used only to encircle the dorsal one-half of the severed trachea.

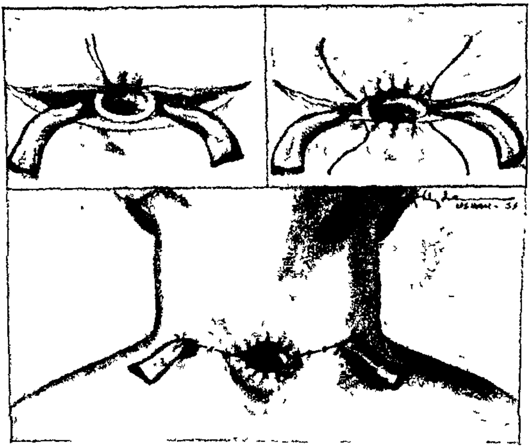

Fig 5 Steps in closure of the low collar incision are illustrated

In a like manner, the lower flap is used to encircle the ventral half of the trachea, again starting by suturing the mid-portion of the incision to the midline of the ventral area of the trachea. The same type of puckering interrupted suture is used. At the sides of the stoma, the two flaps meet and are anchored by interrupted sutures. The drains are pushed to the lateral margins of the incision and sutured in place. The remaining bilateral portions of the incision are closed with deep interrupted catgut sutures (platysma muscle and fascia approximation), and the skin is closed with interrupted black silk sutures.

A No. 9 or 10 tracheotomy tube is then placed in the trachea after the anesthetic intratracheal tube has been removed. This tube is anchored firmly about the lower portion of the neck by hernia tapes. A large pressure dressing is then applied. This dressing should be applied so that all movement of the head and neck is restricted. This step in the procedure is considered extremely important since immobilization is one of the main factors in successful healing.

POSTOPERATIVE CARE.

Postoperative care of patients who have undergone widefield laryngectomy through the low collar incision must be meticulous in order to minimize the formation of sinus and fistulous tracts with accompanying suppuration. The pressure dressing is maintained continuously for at least one week, and is changed and reapplied as necessary during this time. The rubber drains are partially removed starting on the fifth postoperative day and are usually completely removed by the seventh postoperative day. Sutures are removed between the seventh and tenth postoperative days. On the average, feeding by tube is continued for ten days. It may be pointed out that at operation the feeding tube is placed in the lower esophagus, and not the stomach, to avoid postoperative epigastric distress. The usual supportive care plus humidification of the atmosphere and the use of antibiotics is continued postoperatively. When the flaps have healed solidly, on an average of four to six weeks postoperatively, esophageal voice instruction is begun. Usually at this time also the metal cannula can be removed permanently.

SUMMARY.

During the past eight years, experience has shown that the lower collar incision can be used successfully for widefield laryngectomy in the treatment of patients with cancer of the larynx Further experience has shown that its modification (extension of one end of the incision upward) can be used for widefield laryngectomy with primary block dissection. Advantages in its use both to the patient and laryngeal surgeon far outweigh its disadvantages

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ANNOUNCEMENT

The Mount Sinai Hospital, New York, in affiliation with Columbia University announces an intensive postgraduate course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty given by Irving B. Goldman, M.D., and staff, July 14, 1956, to July 28, 1956 Candidates should apply to Registrar for Postgraduate Medical Instruction, The Mount Sinai Hospital, 5th Avenue and 100th Street, New York 29, New York.

THE RECOGNITION AND SURGICAL TREATMENT OF CONGENITAL OSSICULAR DEFECTS.*†

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and

RICHARD A. BUCKINGHAM, M.D.,

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Minor congenital anomalies of the middle ear may cause significant conductive hearing losses and favor the coexistence of middle ear and mastoid disease. The purpose of this presentation is to relate some of our experiences in diagnosing and treating such deformities.

Hearing rehabilitative surgical procedures can be performed in many patients suffering from congenital defects of the middle ear and external canal by using the advanced techniques of temporal bone surgery which were primarily developed for labyrinth fenestration in otosclerosis.

We wish to present four cases of minor congenital aural defects with conductive hearing loss. Three of these cases were proven at surgery to have a synostosis of the incus and malleus as the chief cause of the hearing loss. Before detailed discussion of these cases, we would like to present a working classification of congenital ear defects that has been valuable to us in deciding the indications and type of surgery needed to improve hearing.

Congenital defects of the ear can be catalogued into three clinical groups depending upon the severity of the anomalies.

Class I—Ears with the least abnormalities are listed in this group. The chief features are:

1. Usually normal auricle, or slightly smaller than normal.

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2. Partial, incomplete stenosis of the external auditory canal.

3. Pale eardrum, malleus handle is avascular and appears chalky.

4. Conductive hearing loss which may reach 60 db in the speech frequencies.

5. Mastoid process usually well-developed and pneumatized.

Class II—This group includes the majority of clinical cases of congenital ear defects and is characterized by:

1. Microtia.

2. Complete bony or membranous stenosis of the external auditory canal.

3. Absent drum.

4. Conductive hearing loss usually to 60 db for the speech frequencies.

5. Mastoid process pneumatized.

6. Ossicles normal or deformed.

7. Other abnormalities of a minor nature of the middle ear.

Class III—Ears with the most severe abnormalities are found in this group. Seen in this type of congenital disorders are:

1. Microtia.

2. Complete stenosis.

3. Absent drum.

4. Sclerotic, underdeveloped mastoid process.

5. Severe deformity or absence of the ossicles.

6. Gross deformities or absence of the middle ear.

In 34 patients of all types operated, severe anomalies of the facial nerve pathway have been observed only in this group.

We shall confine our discussion and presentation chiefly to the Class I, or least severe, type of deformities.

For over 20 years, one of us (R H.) has noted in over 2500 cases of temporal bone surgery, that minor anomalies

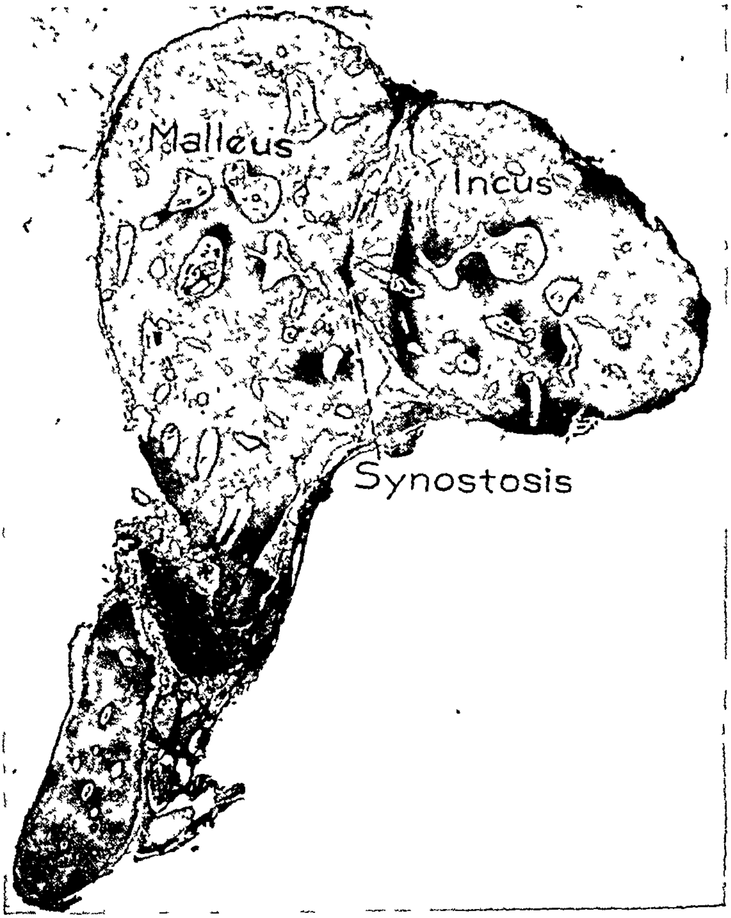


Fig 1 Low power view of synostosis associated with cholesteatoma

of the ossicular chain are often found in association with small attic cholesteatomas. These ossicle anomalies were noted most frequenty in children, as extension of the disease in adults is often severe enough to erode the incudo-malleolar joint.

When care is used to remove the incus and head of the malleus together, and serial histologic sections of the ossicles

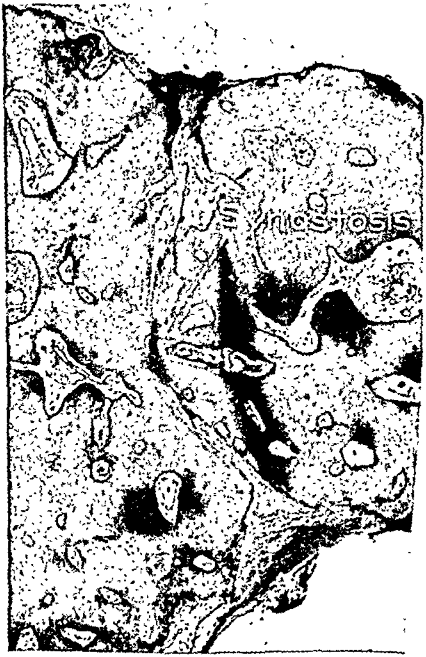


Fig. 2. High power view of Fig. 1, showing bony bridge between malleus and incus.

are made, synostosis and synchondrosis between the incus and malleus are often found (see Figs. 1 and 2). [illegible] [illegible] reported one such instance.

This observation of an apparent developmental anomaly of the ossicles associated with cholesteatoma leads us to believe that in this type of case, both ossicle deformity and cholesteatoma have a congenital etiology. This opinion is fortified by the observation that superimposed otitis media usually results in attic perforation, whereas uncomplicated acute otitis causes central perforation.

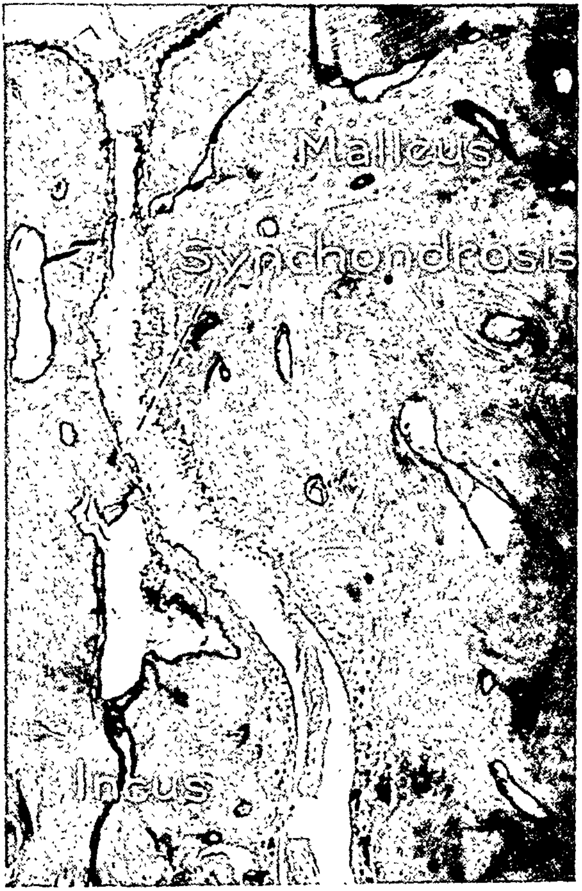


Fig. 3. Case 1. Synchondrosis, high-power view showing cartilaginous union between malleus and incus.

The reported results of other otologists and our own experience has shown that surgery for removal of small cholesteatomas of the attic and antrum is highly successful in eradicating disease and preserving hearing. The cholesteatoma can be removed and further destruction of the conductive apparatus stopped and the hearing often improved over the preoperative level.

Because of these successes in congenital deformity with cholesteatoma and because of our interest in the surgery of major congenital ear deformities, our attention was drawn to those cases of unilateral complete atresia and a partial stenosis of the opposite canal and a bilateral conductive hearing loss. We felt that some of these patients might be deafened in the ear without atresia by the presence of an ossicular impedance When Case 1, reported below, presented himself, we felt justified in attempting surgical exploration and removal of the impedance. We were aware that our results would probably not exceed the 30 db level of an interrupted ossicular chain.

Case 1 D D, a nine year old white male, was first seen at the Illinois Eye and Ear Infirmary in October, 1953 History and examination showed a recurrent left mastoiditis The hearing in the left ear was slightly impaired An extensive simple attico mastoidectomy was performed with good results During the initial examination, it was noted that there was an average 60 db loss in the speech frequencies of the right or "uninvolved" ear

Bone conduction studies revealed normal cochlear reserve bilaterally The right external canal was partially stenosed and presented a constant problem with cerumen accumulation The malleus handle appeared avascular and chalky white The drum was dull There was no fluid in the middle ear on the right X rays showed a normally pneumatized mastoid process

On Oct 7, 1954, an exploratory surgical procedure was performed on the right ear to determine whether the hearing could be improved by interrupting the impeded ossicular chain and at the same time, enlarging the partial stenosis of the external canal

An endaural fenestration type tympano meatal flap was elevated, bony stenosis of the canal removed with the mastoid burr and the lateral attic wall removed A complete mastoidectomy was performed

When the ossicular chain was inspected with the microscope, the incus was found to be smaller than normal and completely fused to the head of the malleus

The incudo stapedial joint was carefully disarticulated and the neck of the malleus severed and the synostotic and synchondritic ossicles removed together (see Figs 3 and 4)

This observation of an apparent developmental anomaly of the ossicles associated with cholesteatoma leads us to believe that in this type of case, both ossicle deformity and cholesteatoma have a congenital etiology. This opinion is fortified by the observation that superimposed otitis media usually results in attic perforation, whereas uncomplicated acute otitis causes central perforation.

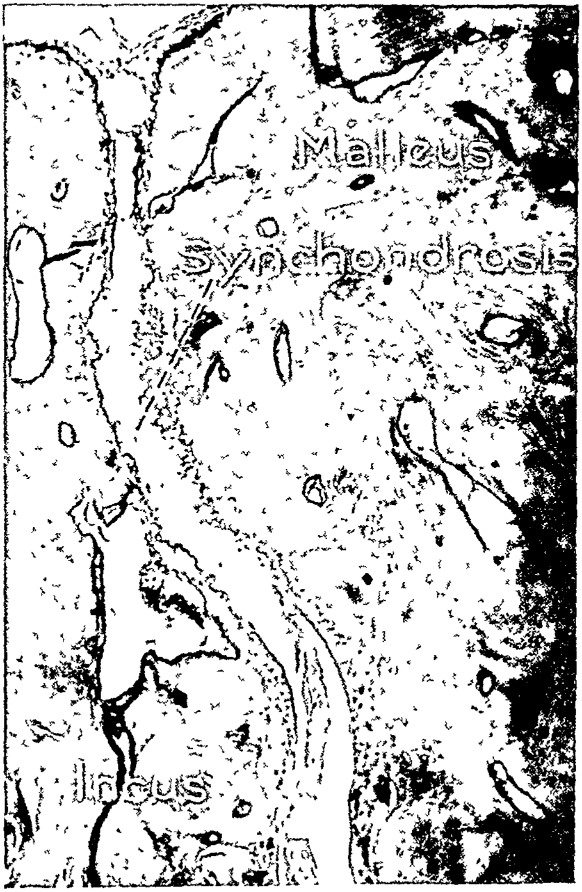


Fig 3 Case 1 Synchondrosis, high-power view showing cartilaginous union between malleus and incus

The tympanomeatal flap was then laid over the excavated mastoid as done in the fenestration operation

The postoperative hearing gain was significant Hearing for the speech frequencies averaged 30 db (see Fig 5) Encouraged by this result, the next case was attempted

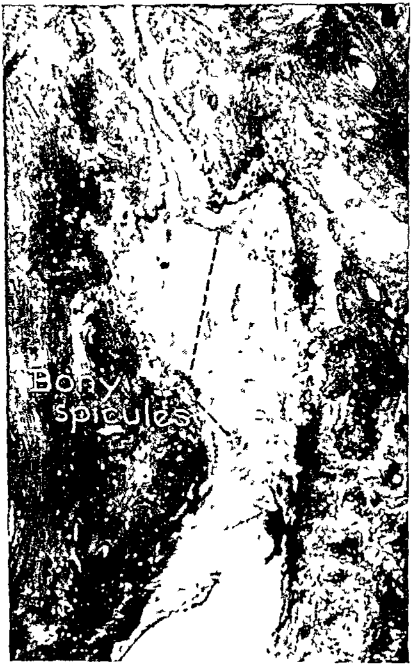


Fig 4 Case 1 Synostosis Mallary stain showing bony spicules between malleus and incus which appears as deeply stained tissue

Case 2 E T, a six year old white male, was referred to the Illinois Eye and Ear Infirmary because of poor speech and hearing Patient was born with multiple congenital defects of the Treacher Collins type and had had multiple corrective plastic procedures to the face and jaws and eyelids In addition to the defects of the face, examination showed partial stenosis of both external auditory canals. The ear drums were normal, except for the typical avascularity of the drums.

Audiometric studies showed an average loss in the speech range of 55 db in the right and 50 db in the left. Bone conduction was normal bilaterally.

Mastoid X-rays showed limited development bilaterally.

A hearing aid was advised, but it was difficult to fit an air conduction aid to the stenotic canals.

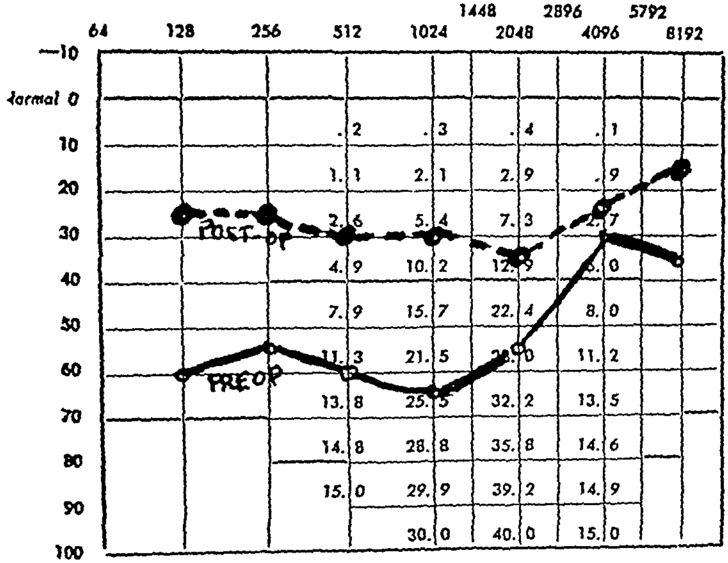


Fig. 5. Audiogram, Case 1, Pre- and post-operative hearing. Dotted line indicates post-op. thresholds.

A modified radical mastoidectomy was performed Nov. 9, 1954, to correct the stenosis of the canal and to explore the middle ear since a Class I type of ossicular deformity was suspected.

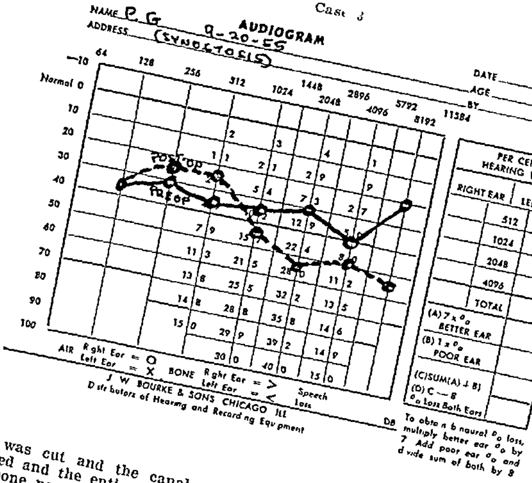
An endaural incision was made and a tympano-meatal flap cut. The bony partial stenosis was removed, the lateral attic wall removed, and a complete mastoidectomy performed. A synostosis of the malleus to the incus was found and the synostotic ossicles removed. The tympano-meatal flap was laid down and packed as usual.

On Jan. 6, 1956, an audiogram showed an average hearing of 30 db for the speech frequencies in the right ear (see Fig. 6).

The next case occurred in the right ear of a patient in whom we had achieved a successful hearing improvement in the left ear which was impaired with a complete atresia of the Class II type.

Case 3 P G, a five year old female, who had been operated on for the above discussed Class II left congenital atresia, was found to have a right hearing loss averaging 35 db This patient had repeated episodes of acute otitis media in this ear which occasioned an additional fluctuating hearing loss The associated narrowing of the external canal and the accumulation of cerumen made it difficult to treat the acute otitis media During an apparent remission of middle ear disease, corrective surgery was undertaken on Sept 20, 1955 At surgery, a tympano meatal flap was cut and the canal was widened The mastoid process was entered and the entire cellular system was filled with granulations, but no bone necrosis was present The lateral attic was removed and the mastoidectomy was enlarged to a complete atticotomy exposing the ossicular chain The incus and malleus were small, deformed, and fused The incudo stapedial joint was separated, and the neck of the malleus clipped, and the incus and head of the malleus were thus removed The flap was then laid in place as in a fenestration operation and the wound packed Her resultant hearing averages 44 db (See Fig 7) This result is confused by the coexisting mastoid disease, however, we do feel that defects which border the practical level of hearing do not warrant interrupting the impeded ossicular chain

Case 3



Case 4 S M, age eight and one half years old, has been observed since May 11, 1953, and presents a very mild Treacher Collins syndrome on the left only This side has aplasia of the temporo mandibular joint, polyotia in the area of the tragus, and a conductive hearing loss on this side averaging 40 db The external canal is mildly stenotic, the drum is dull,

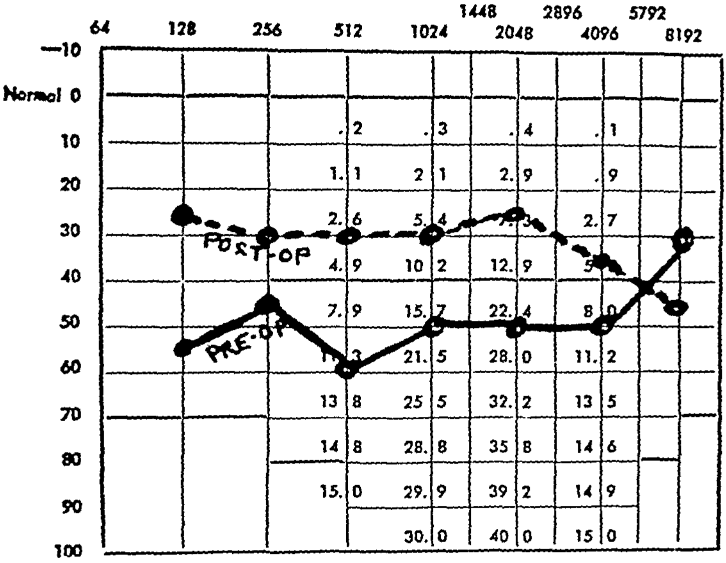


Fig. 6. Audiogram, Case 2, Pre- and post-operative hearing. Dotted line indicates post-op. thresholds.

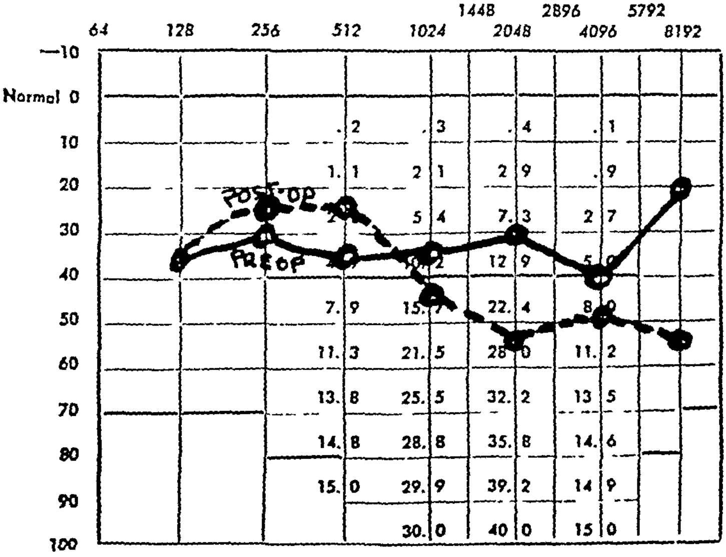


Fig. 7. Audiogram, Case 3, Pre- and post-operative hearing. Dotted line indicates post-op. thresholds.

and the hammer handle is avascular. Because of the associated congenital deformities, we suspect the hearing loss in this ear to be the result of impedance of the ossicular chain, probably due to synostosis; however, because of the completely normal hearing on the right ear and small expected gain from surgery, we have not operated this patient

DISCUSSION.

Congenital synchondrosis and synostosis of the ossicular chain are regularly seen associated with stenosis of the external auditory canal. H. Marks,[2] in a survey of the literature, found that in forty-eight reported cases of congenital stenosis of the external canal, only two ears had normal adult ossicles. F. Altman has reported ossicle deformities in almost all of the severely congenitally deformed ears that he has studied.

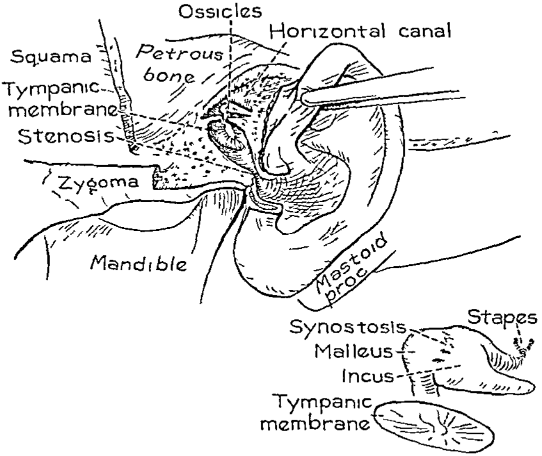


Fig 8 Diagrammatic sketch showing stenosed external canal associated with synostosis of ossicles

In our own thirty-four cases, only three patients had adult-appearing and mobile-enough ossicles to merit non-removal at the time of surgery.

We wish to point out that the ossicular deformities may be present alone or in association with mild stenosis of the external canal and cause handicapping hearing losses that may be improved significantly (see Fig. 8).

Such isolated anomalies are rare, but careful search for the diagnostic criteria presented should reveal more cases of this type. H. House[3] recently reported two cases of fusion of the incus to the tympanic ring as the only apparent defect of the complex conductive hearing mechanism, and excellent hearing improvement resulted after freeing the impedance of the ossicular chain.

SUMMARY.

1. Minor congenital ossicle defects of synostosis and synchondrosis of incus and malleus may cause severe conductive hearing loss.

2. A classification of congenital aural defects is presented, and attention is drawn to the common association of cholesteatoma and ossicular defects.

3. Three proven cases of synostosis of the malleus and incus as the sole cause of conductive hearing loss and surgical correction are presented. A fourth probable case is presented.

4. Minor congenital ossicular anomalies may cause severe conductive hearing losses; these ears may be microtic; the canal may be mildly stenotic; the drum appears dull, and the hammer handle of the malleus appears chalk-like and avascular.

5. This type of anomaly is probably more common than is realized. Careful search should find more cases of this type.

6. Altman[4] in his excellent report of a case of congenital atresia of the ear observed an osseous connection between the stapes and the facial canal. He stated that the chances for improvement of hearing are doubtful due to the frequency of bony bridges between the stapes and the medial tympanic wall and other minute deformities which might escape detection While the results do not always meet our expectations, meticulous technique, the use of the operating microscope, and newer Cortisone-like drugs which decrease inflammation and scar tissue, encourage us to continue in our efforts to improve surgically the congenital conductive hearing loss patient

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DR CHARLES BRAMMAN MEDING AWARD

On May 21, 1956, at The Harlem Eye and Ear Hospital, Dr Eugen Grabscheid received the Dr Charles Bramman Meding Award This award, granted annually in memory of Dr Meding, was bestowed upon Dr Grabscheid in recognition of his outstanding clinical service at The Harlem Eye and Ear Hospital and for his presentation of programs of study in the interest of the advancement of the art of Otolaryngology

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UNIVERSITY OF ILLINOIS, COLLEGE OF MEDICINE

The next Laryngology and Bronchoesophagology Course to be given by the University of Illinois, College of Medicine, is scheduled for the period November 5 through November 17, 1956 The course is under the direction of Dr Paul H Holinger

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois, College of Medicine, 1853 W Polk Street, Chicago 12, Ill

SURGICAL TECHNIQUE OF REMOVAL OF INFRATEMPORAL MENINGIOMA.

JOHN J. CONLEY, M.D.,*

GEORGE T. PACK, M.D.,*

and

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New York, N. Y.

Meningiomas causing exophthalmos from their intra-orbital placement may originate from several sources, *viz.*, *a.* anterior cerebral meninges, *b.* meningiomas of olfactory groove passing through the foraminal narrows (Cushing), *c.* meningeal tumors lying within and expanding the vaginal sheath, *d.* sphenoidal ridge tumors growing into the orbit and *e.* meningiomas producing tumor-hyperostosis from invasion of the orbital bones. Those meningiomas not arising from the sheath of Schwalbe and not involving the optic nerve proper with atrophy and blindness usually are located in the lateral orbital compartment. Cushing[1] directed attention to the tendency of meningiomas to expand in the line of least resistance and "to crowd their way into all neighboring anatomical crannies and pockets."

In the patient who is the subject of this case report, the meningioma presented itself chiefly in the infratemporal fossa, having extended there from the left lateral orbit. The lateral wall of the bony orbit was invaded by the tumor. The optic nerve and sheath of Schwalbe were grossly free of evidence of tumor. Psammoma bodies within the tumor aided in the identification of its histogenesis. The histological findings in these extracranial meningiomas are consistent with the hypothesis that all of them may be intracranial in origin even though the bulk of the tumor is without the skull. In this

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patient, there are no symptoms, no physical findings and no radiographic evidence of intracranial involvement, but the disease will be suspected until a long period of postoperative observation furnishes convincing proof otherwise.

CLINICAL HISTORY

H M, a 45 year old white male, related that he had been aware of a gradual swelling in the region of the left temple and increasing prominence of the left eye for two months prior to his first visit to this clinic on April 7, 1955 There were no symptoms related to this mass

Clinical examination revealed a deeply fixed mass completely filling the left infratemporal fossa and protruding in the region of the temple just above the left zygoma The left eyeball was proptosed 2 mm, but vision, extra ocular movements, and fundoscopic examination were normal There was no trismus, no cranial nerve involvement, and X rays of the skull with particular attention to the infratemporal fossa and orbital regions were unrevealing

Aspiration biopsy was interpreted as a probable malignant tumor in the infratemporal fossa and retro orbital region Lymphoma, primary somatic tumor, neurogenic tumor and tumor of the lacrimal gland were also considered

Because of the indefinite histogenetic diagnosis afforded by the aspiration biopsy and the clinical features simulating the Mikulicz lymphoma syndrome, a therapeutic test by X ray therapy was started A tumor dose of 4200 r was given during three weeks of irradiation using the following factors 250 KV, 50 cm T S distance, 4 cm cone, 2 ports, filter of 1 5 mm Cu The regression was not typical of lymphoma within the customary period of post irradiation observation therefore, surgical exploration was advised

TECHNIQUE.

The skin incision began in the medial part of the left eyebrow, curving inferiorly and laterally to a position just above the zygoma and then posteriorly to the superior attachment of the ear. Here it joined a vertical excision extending from the region of the temple down to the level of the tragus The skin flaps were elevated widely down to the superficial temporal fascia, thus affording an excellent view of the lateral bony orbit, zygoma and temporal muscle. The zygomatic bone was completely exposed along with the zygomatic extension of the temporal bone by cutting the origins of the masseter, quadratus labii superior, and zygomaticus muscles. The temporal segment of the zygomatic arch was green-stick fractured posteriorly. The remainder of the overhanging zygomatic bone was removed with appropriate bone-cutting rongeurs. A firm fixed mass could be felt filling the entire infratemporal fossa and causing a bulge in the temporal muscle at this level. The temporal muscle was then transected 2 cm. above the coranoid process of the mandible, and the

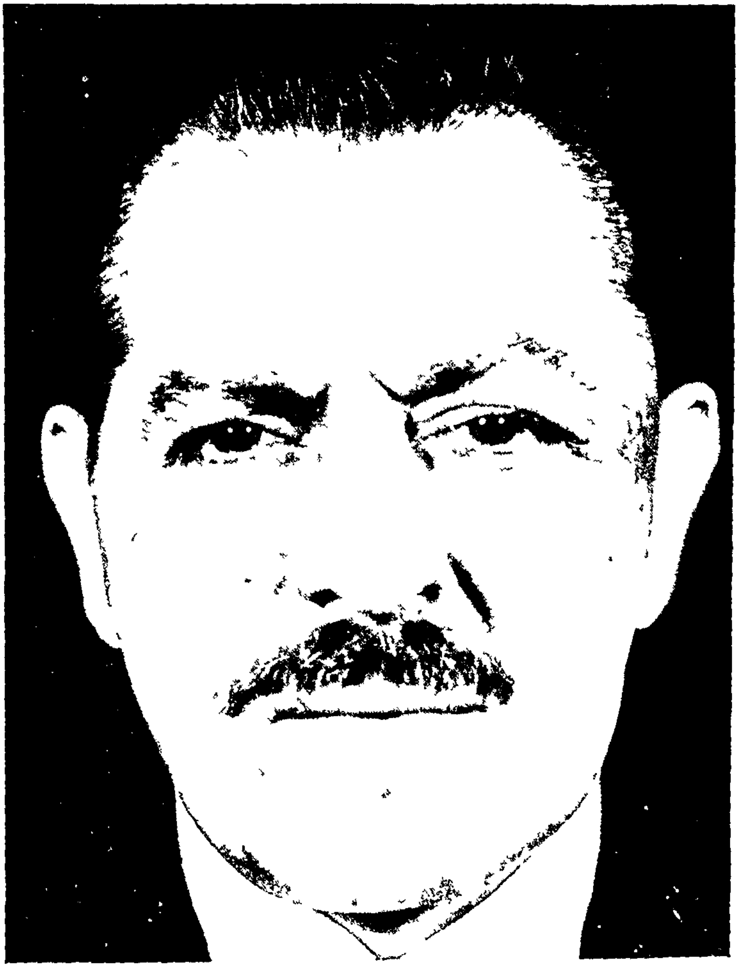

Fig 1-A Preoperative view showing protuberance in left infratemporal area and post-irradiation pigmentation

tumor came into direct view. It contained an indefinite capsule and extended in the direction of the infra-orbital fissure and orbital cavity. The zygomatic portion of the lateral bony orbit was then totally removed, thus uncapping the infra orbital fissure and establishing a common exposure of the orbit and infratemporal fossa The orbital portion of the tumor was

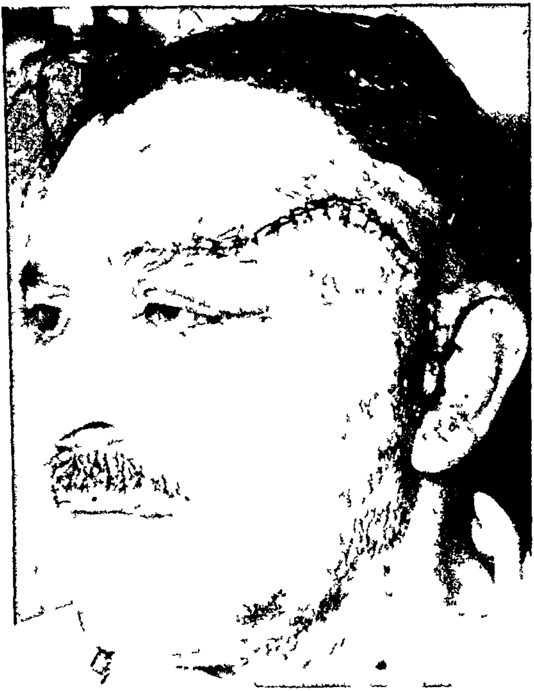

Fig 1 B One week postoperative

attached to the infra-orbital fissure From the primary attachment in the region of the infra-orbital fissure, the tumor extended medially and posteriorly over the great wing of the sphenoid A small section of bone at this site was destroyed by tumor. Once the exposure was complete, the tumor came out with surprisingly little difficulty. The maxillary nerve was resected, and the internal maxillary artery was ligated.

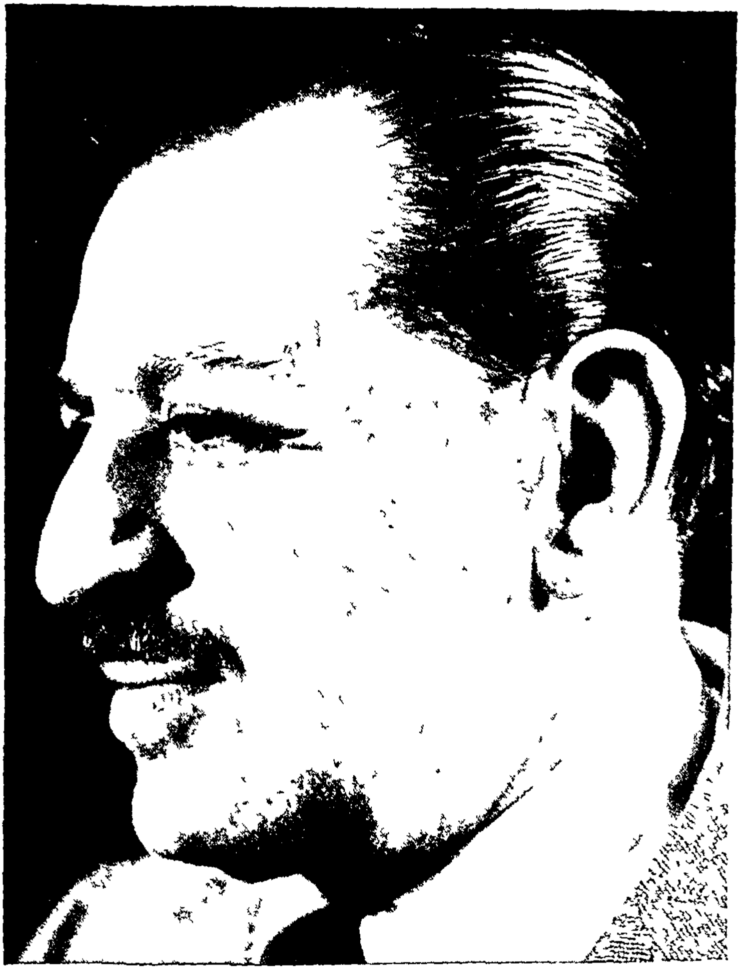

Fig 1-C Three months postoperative

A portion of the pterygoid wing of the sphenoid was removed, and the dura appeared normal. The wound was then closed in layers by first approximating the cut segments of the temporal muscle and the subcutaneous tissue with fine silk. The

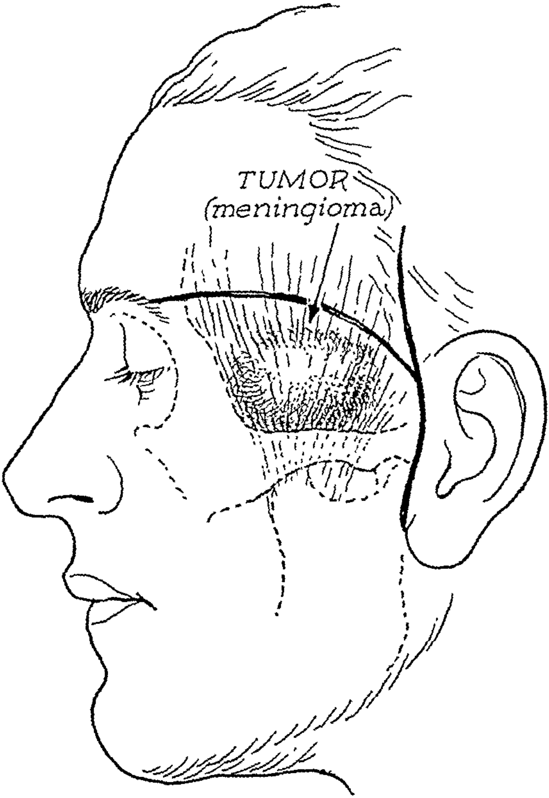

Fig 2. Meningioma situated in the infratemporal space.

residuum of the zygomatic portion of the temporal bone was placed into position and the skin flaps closed with silk, with a smaller rubber drain extending into the temporal fossa.

The postoperative course was uncomplicated. The patient complained of a left-sided headache for two days postoperatively, which required mild analgesia. The periorbital edema subsided gradually within two weeks. The extra-ocular movements of the eyeball were normal. There was no diplopia and no proptosis. There was an area of anesthesia in the region of the infra-orbital nerve. Mastication was slightly weakened on the left side but was adequate. The drain was removed on the third postoperative day, and the wound healed per primum (see Figs. 1 and 2).

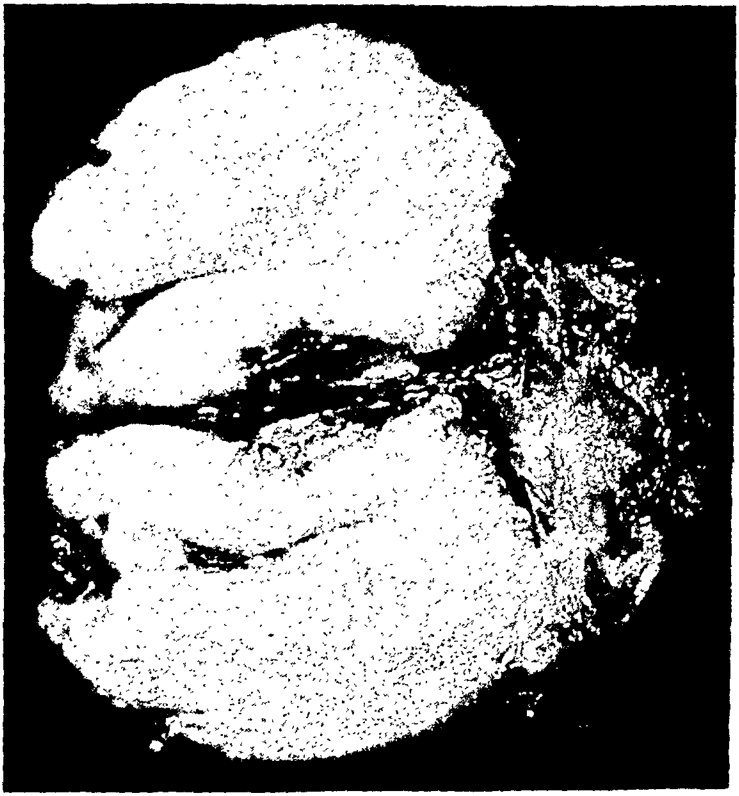

Fig. 3. The tumor of the temporal fossa is homogeneous in appearance.

MICROSCOPY.

The tumor measured 4 x 3 x 2 cm. and was lobulated, pink-gray, and moderately firm. The cut surface was dense, homogeneous, and tan-gray (see Fig. 3).

The tumor was characterized by a whorled pattern of fibrillary cells of homogeneous appearance. The whorls were separated by relatively narrow bands of hyaline connective tissue. The nuclei were large and oval. Occasionally, individual nuclei were large and deeply stained. Mitoses were absent (see Fig. 4). At the periphery, the tumor tissue penetrated into the adjacent muscle. At this site, the nuclear

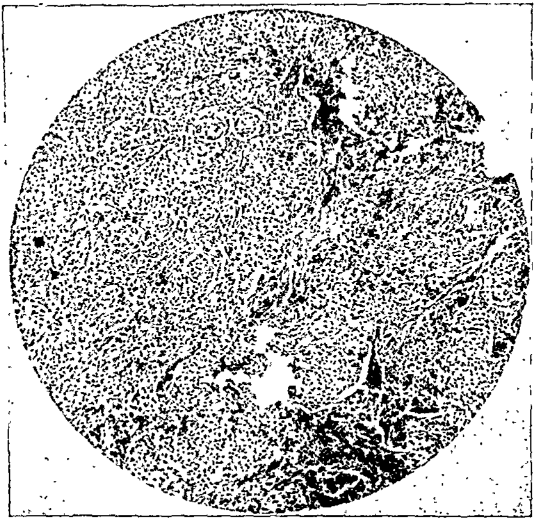

Fig. 4. The fibrillary masses are separated by bands of fibrous tissue producing a lobulated pattern. The masses have a homogeneous appearance and the nuclei are large and oval. X 60.

pattern displayed more anisocytosis than the main mass. The vessels were dilated and small, edematous foci were numerous.

The marrow spaces of the temporal bone were filled with tumor tissue of similar nature. In addition, there were several Psammoma bodies (see Fig. 5). The specimen was diagnosed as invasive meningioma.

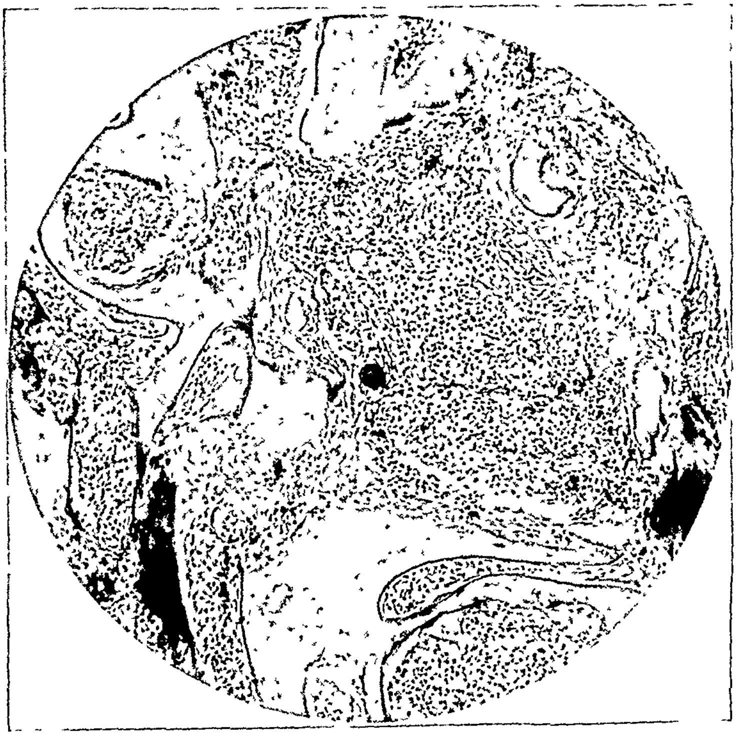

Fig. 5. The marrow spaces are filled by tumor tissue of whorled pattern. A psammoma body is seen in one area. X 90.

SUMMARY.

An unusual case of extracranial meningioma involving the infratemporal and orbital regions was removed by a direct approach to the infratemporal space. The direct approach gave better exposure of this region than the usual scalp flap.

The eye was not affected and the postoperative course was uneventful.

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139 East 36th Street.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Arrangements have been completed for the joint meeting of the North Carolina Society of Eye, Ear, Nose, and Throat, and the South Carolina Society of Ophthalmology and Otolaryngology September 17, 18, 19, 1956. Headquarters will be the George Vanderbilt Hotel, Asheville, North Carolina.

An unusually attractive program has been arranged, and a large attendance is anticipated.

Asheville, North Carolina, is in the mountains of Western North Carolina, and is a particularly beautiful spot in this season of the year.

For further information write Roderick Macdonald, M.D., Sec. and Treas., 330 East Main Street, Rock Hill, S. C.

WASHINGTON UNIVERSITY, SAINT LOUIS DEPARTMENT OF OTOLARYNGOLOGY.

Offers a Basic Science Course in Otolaryngology to start Monday, September 17, 1956. Complete information about the course may be obtained by writing to Theo. E. Walsh, M.D., Head of the Department of Otolaryngology, 640 S. Kingshighway, St. Louis 10, Missouri.

RELATIONSHIP BETWEEN ETIOLOGY OF HEARING LOSS AND RESULTANT AUDIOMETRIC PATTERN.*

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and

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Nashville, Tenn.

THE PLAN OF STUDY.

The Origin of the Cases.

The original data for this study was derived from the pure tone audiograms of 175 hearing impaired children in the age range from six to 14 years, all of whom had been referred to the Hearing and Speech Center† for Audiological Evaluation during the 21 month period ending April 1, 1953. Because of meager information, either in the medical history or the informant's report in the initial interview, 24 audiograms were discarded, leaving a total of 151 for detailed study and analysis.

Each of these 151 children was referred to the diagnostic clinic by an ear, nose, and throat specialist, with an accompanying otological report.

The audiological assessment at the Center consisted of detailed hearing testing, comprising both pure tone and speech audiometry, complete case history, and testing in allied areas, including intelligence screening, when indicated. Each pure tone audiogram was done by a trained audiologist in a sound proof testing chamber. For the pure tone testing, four different commercial audiometers were used, but each was methodically maintained in calibration with reference to average normal threshold.

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* Read at the Meeting of the Southern Section of the American Laryngological, Rhinological and Otological Society, Inc., Houston, Tex., Jan. 27-28, 1956.

† Research work of the Staff of the Bill Wilkerson Hearing and Speech Center.

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THE METHOD OF CLASSIFYING AUDIOGRAMS.

In any study involving the grouping together of a large number of audiograms, it is essential that they be classified in a definite and simple manner. Several methods have been contrived in the past 20 years. One of the earlier was devised by Guild[1] in 1932. His emphasis was on the main characteristics of the plotted line; that is, the more general form, direction, and position.

In the interest of the United States Public Health Service, Ciocco[2] in the early thirties, began a survey of the hearing of almost 1400 school children. Ciocco's method of classification included eight basic categories.

A more recent method was devised by Carhart[3] to meet the needs of the acoustic clinic at the Deshon Army Hospital during World War II. His aim was to achieve a coding procedure which would be sufficiently inclusive in scope and universal in application.

The categories of audiogram patterns devised for the present study were not based on any one previous method, but salient features from each of the methods suggested by Ciocco and Carhart were adapted and integrated into the final plan. The outcome was the following eight classifications, which seemed to provide for the various deviations encountered in the audiograms, and at the same time afforded the simplicity of description.shown by Ciocco and the definitive scope of Carhart:

Group 1. Good hearing for all tones. All tones heard at an intensity equal to or less than 20 db at six of the seven frequencies tested.

Group 2. Near normal hearing at the two lower octaves of the speech frequency range with precipitous drop in acuity at 2,000 cps. or above.

Group 3. Gradual downward slope with progressively greater loss for higher frequencies at a slope of five to ten db per octave across the audiogram.

Group 4. Marked high tones loss, characterized by at least one 20-db slope per octave; within the 250 to 2,000 cps. range, or by no hearing within the audiometer limits for at least two of the frequencies above 1,000 cps.

Group 5. Moderate loss for all tones; thresholds between 20 and 65 db for at least six of the seven frequencies of the audiogram.

Group 6. Marked loss for all tones; thresholds between 65 and 100 db for at least four of the five middle frequencies (250 to 4,000 cps.).

Group 7. Rising audiometric contour with general pattern of loss for frequencies below 1,000 cps. ten to 30 db greater than for tones above 1,000 cps.

Group 8. Trough-shaped curve with greatest depression occurring in speech frequency range.

THE METHOD OF CLASSIFYING ETIOLOGIES.

Since the initial categorization was based on the audiometric curve, the various etiologies determined for each subject's hearing loss were at first merely listed audiometric pattern groups. In order to tabulate data for inspection and analysis, it soon became evident that the causative factors, like the audiogram curve, needed to be classified into the smallest number of categories feasible.

This problem presented some difficulty because of the many known causes of hearing impairment. The chief source of information for determining the etiology was the otological examination and the case history. When no cause whatever could be found, the etiology was noted as "cause undetermined."

Otitis media and hypertrophied tonsils and adenoids were combined to form one group, since these conditions are somewhat alike in their initial effects upon the hearing mechanism. Hypertrophied tonsils and adenoids, and otitis media cause a fairly uniform reduction in acuity for all tones.

Such diseases as influenza, meningitis, pneumonia, mumps, measles, virus, and whooping cough may involve the VIIIth nerve or its terminal branches. In view of this fact, they lend themselves to a single classification, which Fowler[4] has spoken of as inflammatory neuritis.

Loss of hearing due to heredity, birth injury, rubella, prematurity, drugs, and head injury is known to cause perceptive hearing loss. Still it was felt that for purposes of this study they needed separate classification, because each condition may have a selective action on certain segments of the auditory neural mechanism. Unfortunately, the number comprising these groups was in several instances quite small.

The classification of psychological etiology was used when marked discrepancy between the speech and pure tone results was found to exist. These cases also evidenced functional hearing within normal limits, although the pure tone audiometric evaluation indicated a decrement in acuity. The evidence of normal hearing was in each instance established conclusively by the use of speech audiometry.

TABLE I.

INCIDENCE OF ETIOLOGIES OF HEARING LOSS ACCORDING TO AUDIOMETRIC PATTERN.

	Audiometric Pattern Groups								
Etiology	I	II	III	IV	V	VI	VII	VIII	Total
Otitis Media; Infected T. & A.	21	2	6	3	16	3	15	2	68
Inflammatory Neuritis	3	2	5	6	4	3	2	5	30
Heredity	0	0	2	3	5	2	1	1	14
Birth Injury	0	0	2	8	0	0	0	0	10
Psychological	0	0	1	0	4	1	3	0	9
Cause Undetermined	1	3	0	2	0	1	0	0	7
Rubella	0	0	1	0	2	1	0	2	6
Prematurity	0	1	0	2	1	0	0	1	5
Drugs	0	0	0	0	0	1	0	0	1
Head Injury	1	0	0	0	0	0	0	0	1
Total	26	8	17	24	32	12	21	11	151

The incidence of the various causes of hearing loss in the different audiometric pattern groups in shown in Table I. The information presented in this Table forms the basis for the detailed analysis of audiogram patterns in this study.

INFORMATION YIELDED BY AUDIOGRAM PATTERN GROUPING.

Group I. Good Hearing for All Tones. Fig. 1 represents the mean air and bone conduction curves for the 26 children in the Group I category, which includes good hearing for all tones with hearing as good as 20 db for six of the seven frequencies tested in each individual case.

Twenty-one, or 80.8 per cent of the 26 cases were thought to have this slight hearing decrement as a result of either otitis media or infected tonsils and adenoids. In a number of instances the children in Group I may have received otological care which improved their hearing status prior to the audiological testing at the clinic. This situation occurred with seven of the 26 cases in the group.

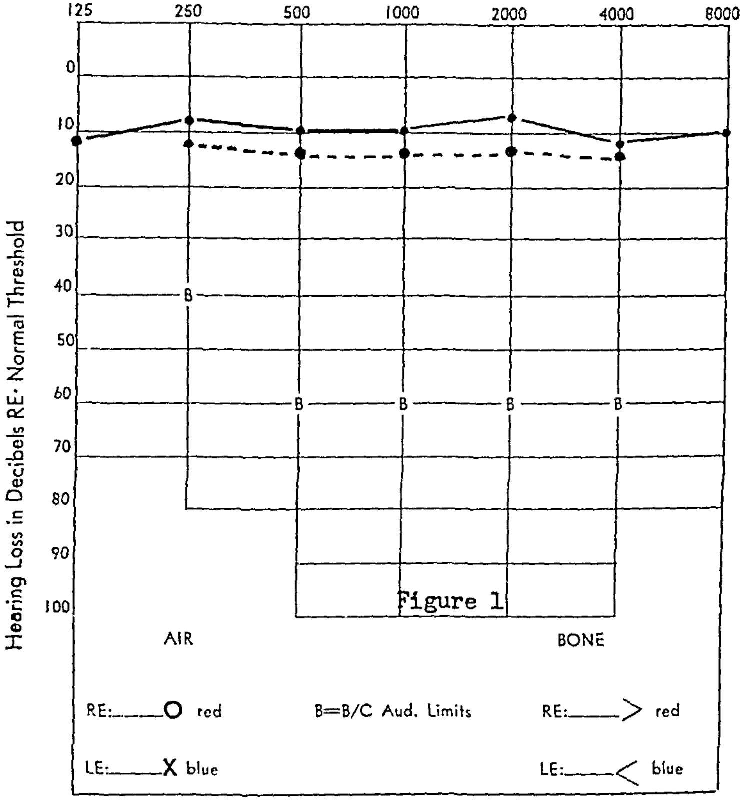

Figure 1

The fact that as many as four of each five audiograms in Group I were associated with an etiology of otitis media, or hypertrophied or diseased tonsils and adenoids, is considered significant. The mildly depressed air conduction curve throughout the frequency range is the type of hearing pattern which beginning middle ear difficulty may often be expected to present.

Group II. Near Normal Hearing in Low Frequencies with Sharp Drop-off in High Frequencies. Only eight of the total number of audiograms analyzed fell in the Group II configuration, which indicates near normal hearing at the 500 and 1,000 cycle tones, but with a precipitous drop in acuity beginning at 2,000 cps. or above. Persons with this type of hearing loss, represented audiometrically by the mean curve in Fig. 2, were unable to hear many of the high frequency consonant sounds, such as (s), (sh), and (th) in everyday conversational speech.

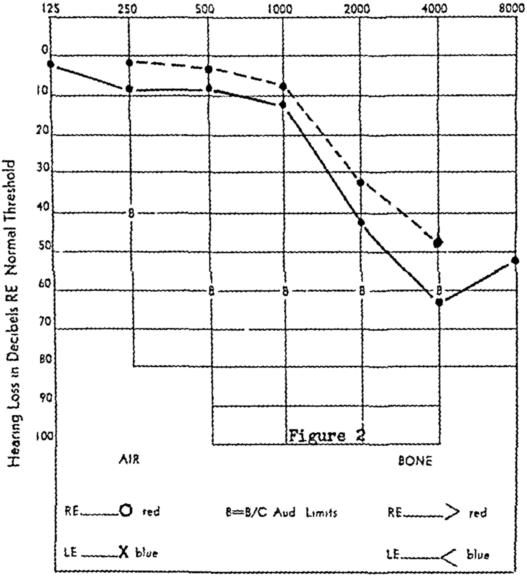

Figure 2

It is noted that the eight included in this classification were rather randomly distributed as to the causative factor. For three, or 34.5 per cent, no cause could be determined.

The closely parallel relation between the mean bone and air curves, as shown in Fig. 2, would seem to indicate that *this type of pattern is the result of nerve damage to particular segments of the basilar membrane.* The random distribution of etiologies and the small number in this group make any further statement as to the cause of this type of hearing impairment unwarranted.

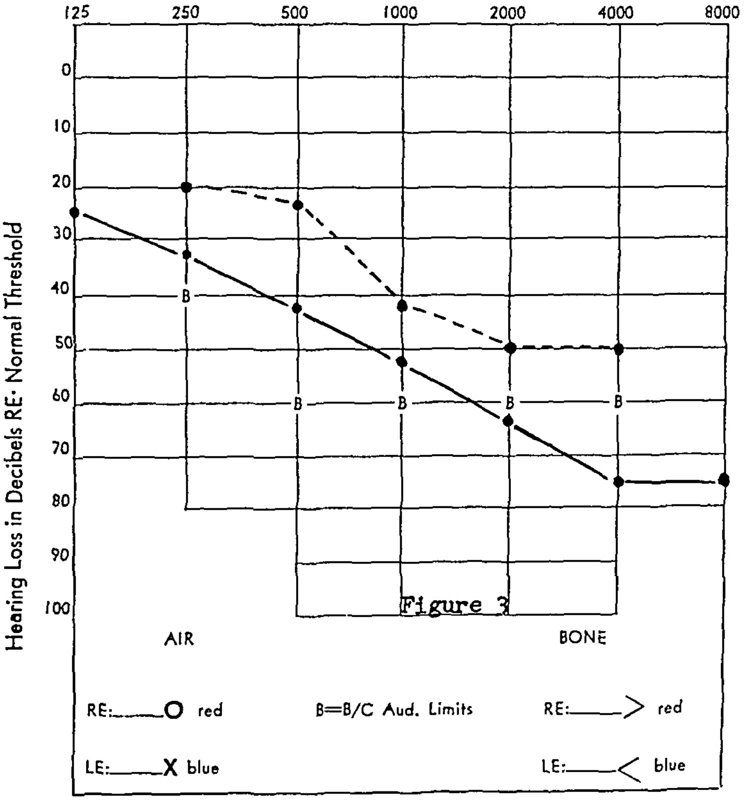

Figure 3

Group III. Gradual High Tone Loss. The configuration revealed by Group III, presented in Fig. 3, denotes a serious hearing problem, with the gradual downward slope increasing about ten db per octave from left to right. The loss in acuity throughout the speech frequency range denotes an inability to hear most of the consonant sounds in conversational speech. Seventeen children, or 11.2 per cent of all the cases, were in this category. Six of 17, or 34.3 per cent of the group, were associated with otitis media, or infected or enlarged tonsils and adenoids. These findings supported the fact that chronic middle ear conditions often may eventually result in a mixed-type loss which is irreversible in large part because of the extent of perceptive impairment. The slight conductive component of the mean audiogram is contributed chiefly by the six mentioned above. Five, or 29.4 per cent, were associated with inflammatory neuritis from mumps, measles, whooping cough, and meningitis. Inspection of their individual audiograms showed that these cases were all of the perceptive type. Two birth injury, two heredity, one rubella, and one psychological were the other etiologies in this pattern.

Group IV. Marked High Tone Loss. The mean audiometric curve for Group IV, seen in Fig. 4, presented a marked downward slope within the speech frequency range, with a severe depression in the higher frequencies. Each audiogram chosen with this pattern had at least one per-octave drop as great as 20 db in the 250 to 2,000 cps. octave range. The mean air conduction threshold for 1,000 cps. was 25.4 db greater than for 500 cps. This octave band appeared to be the one which is most often characterized by this essential feature of the marked high tone loss audiogram.

This type of pattern was evidenced by 24, or 15.9 per cent of the total 151 audiograms, and was, therefore, the third largest group numerically. Eight children, representing one-third of the group, gave a history of birth injury, suggesting that this etiology has a selective effect upon the acuity for high frequency sounds.

(Twelve additional cases of birth injury were studied after the completion of this paper. Ten of these cases had a hearing loss of less than 20 db at 125, 250 and 500 cps., with a sharp drop for the higher tones.) The auditory acuity of the other two cases followed the pattern shown in Fig. 4. Six children, or one-fourth of the number in the group, had hearing loss attributed to inflammatory neuritis associated with meningitis and childhood exanthemata. Two or three each were distributed randomly among all the etiologies except rubella, psychogenic, drugs, and head injury.

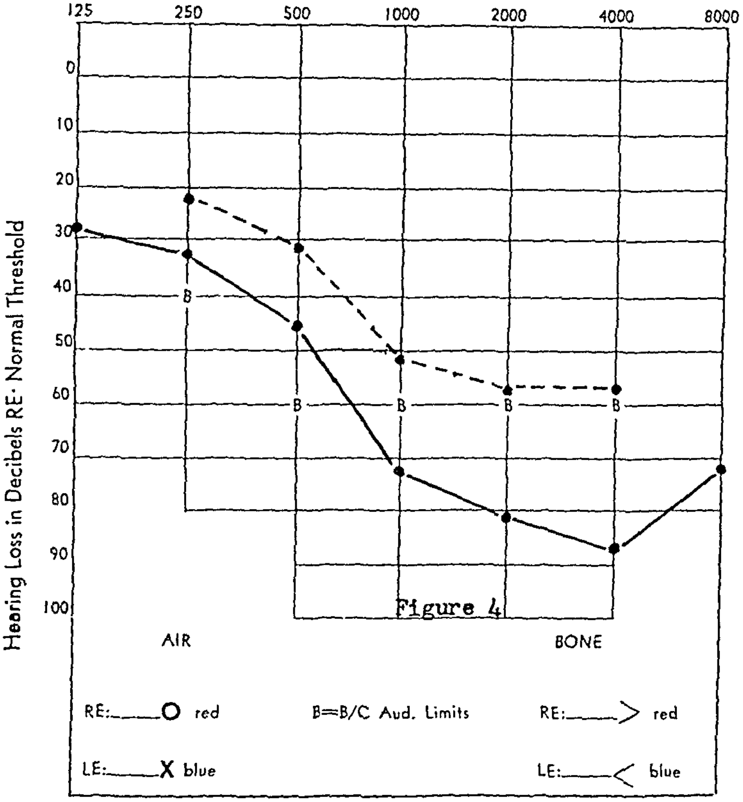

Figure 4

Group V. Moderate Loss for All Tones. The audiograms making up Group V represented a moderate loss for all tones. Thirty-two, or 21.2 per cent of the total number of audiograms studied constituted this category, which comprised the largest numerical group of the eight major audiogram pattern classifications. Fig. 5 revealed a fairly uniform loss throughout the frequency range, with all mean thresholds except that for 125 cps., lying between 40 and 50 db. It was significant to note that 16, or 50 per cent of the subjects, in this group, were referred to the Hearing Center with otitis media or infected or enlarged tonsils and adenoids. This fact accounted for the substantial conductive component shown by the mean audiogram air-bone gap. Seven, or 21.9 per cent, of these cases were diagnosed as either rubella or heredity. The incidence of these conditions is known to be reflected audiometrically by perceptive type involvement, which would be the factor that would depress the bone conduction curve in Fig. 5.

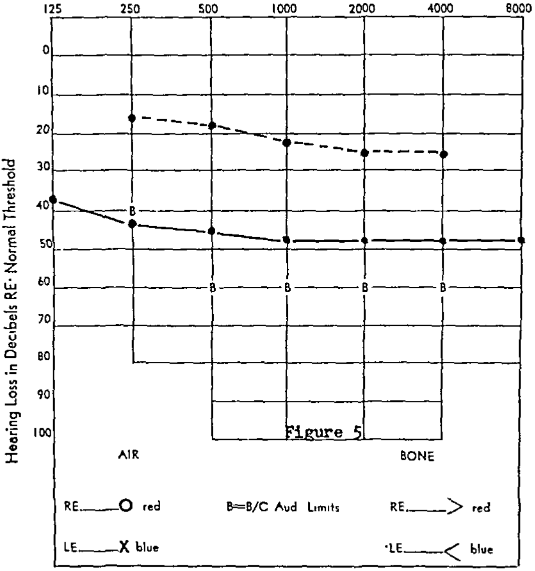

Figure 5

Group VI. Marked Loss for All Tones. Group VI was represented by 12, or 7.9 per cent of the total number of audiograms studied. This pattern was characterized by marked loss for all tones. Children in the group are classified as profoundly deaf. *Because bone conduction testing has a 60 db* intensity limit, the bone conduction curve was falsely weighted with those subjects having better hearing than 60 db for bone conduction tones. In order to include these with poorer hearing, thresholds were recorded at 60 db for all cases for whom the hearing level was actually poorer, but where true thresholds could not be obtained because of the intensity limits of the audiometer. This limitation accounts for the fact that all the mean bone conduction curves for the study show mean threshold values which indicate somewhat more conductive component than might be expected.

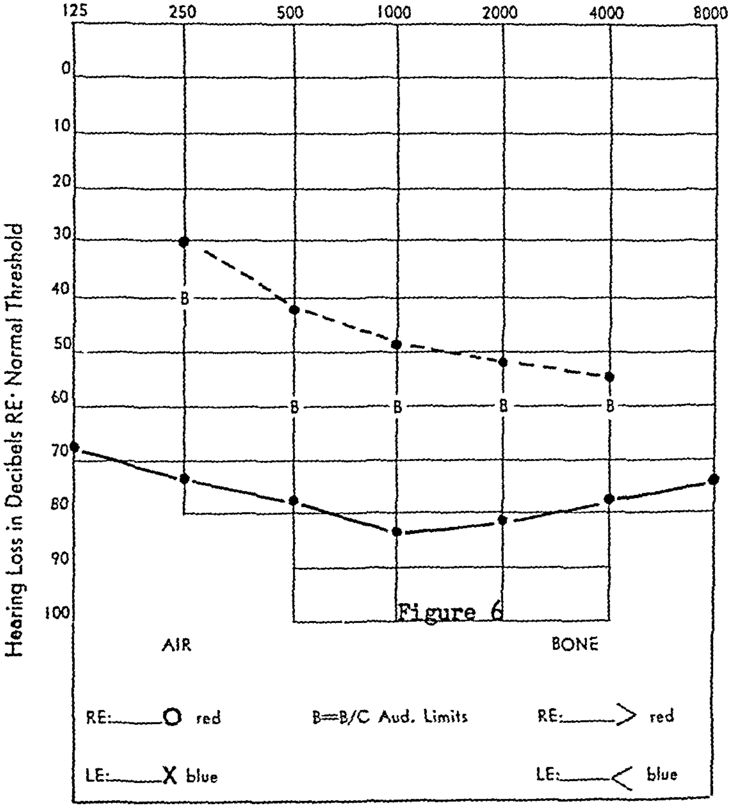

Figure 6

The 12 cases are rather randomly distributed throughout all the etiologies, except for prematurity, birth injury, and head injury. Three cases each, making one-half the total, were associated with the otitis media and the inflammatory neuritis causes.

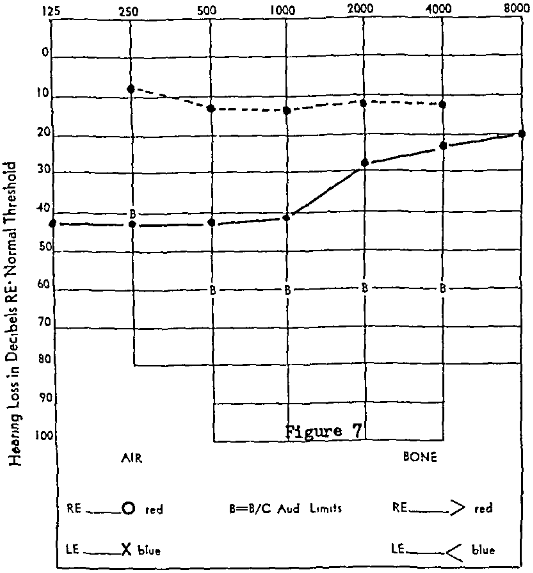

Figure 7

Group VII. Rising Audiometric Contour. In Group VII were found 21, or 13.9 per cent of the audiograms from the total number. This pattern was characterized by the rising audiometric contour, exactly opposite to the gradually downward slope denoting Group III. This type of profile rose gradually from 1,000 to 8,000 cps., with usually more uniform loss of hearing for the frequencies below 1,000 cps. (see Fig. 7). It was considered significant that 15, or 71.4 per cent of the audiograms displaying this pattern were associated with an etiology of otitis media and/or infected or enlarged tonsils and adenoids. The presence of the nearly maximum air-bone gap shown by the mean audiogram, indicated that this pattern may be associated with the purely conductive type lesion. This is in agreement with what is known about the effects of the etiology which was so prominent in this group. The incidence of this pattern was noted once in association with heredity, and twice with inflammatory neuritis. These three audiograms may account for the mild bone conduction depression, since they were generally conceded to cause a nerve-type deafness. Three, or 14.3 per cent, were of psychological etiology. The child with functional loss may well be expected to display relatively greater inability to hear low frequency sounds, since they comprise the major portion of environmental sound.

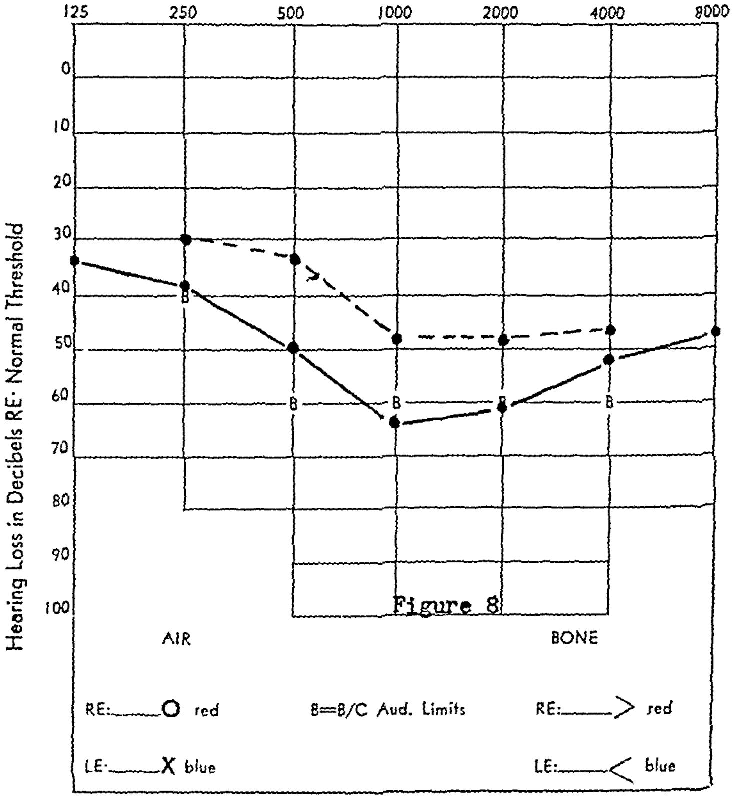

Figure 8

Group VIII. Trough-Shaped Curve. Eleven, or 7.2 per cent, of the total number of audiograms studied presented a trough-shaped curve, with the greatest depression occurring within the speech frequency range. Examination (see Fig. 8) showed that the higher frequencies above 1,000 cps. were affected more than those below 1,000 cps. This perceptive symptom was reasonable, in view of the fact that the largest percentages occurred in the rubella and inflammatory neuritis categories, being 18.2 and 45.5 per cent, respectively, since these two etiologies characteristically presented a nerve type loss. In six of the audiograms the point of greatest severity of loss was found to be at 1,000 cps., indicating this frequency may be the focus for the greatest damage in certain pathologies of hearing. The upper limit of the trough was at 20 db, while the point of greatest depression in any audiogram within the group occurred at 80 db. In nine of the 11 audiograms, the entire trough depression was centered in the speech frequency range, with thresholds occurring between 45 and 75 db.

INFORMATION YIELDED BY ETIOLOGIC GROUPS.

As stated previously, classification of data for the present study included a consideration of two factors, audiometric contour and etiology of hearing loss; therefore, it was necessary in a thorough analysis of the data to consider not only how the different etiologies were included in each of the eight patterns, but also how the different patterns were distributed among the ten etiological groups.

Sixty-eight, or 45 per cent of the 151 cases were diagnosed as otitis media or enlarged and diseased tonsils and adenoids. It was felt that this group was large enough to utilize the chi-square technique to determine the significance of the observed frequency distribution among the eight audiometric patterns. The chi-square value obtained was 41.312 which meets the one per cent level of confidence, indicating that the observed distribution of cases in this etiological group could not have occurred by chance factors alone, if there had not been some relationship between etiology and resultant audiometric contour.

It would appear that middle ear impairments caused by otitis media and enlarged tonsils and adenoids may be significantly associated with both the "moderate loss for all tones" and the "slightly rising audiometric contour" audiograms.

The next largest etiological category was that of inflammatory neuritis, *consisting of 30, or 19.9 per cent of the 151* cases distributed among the eight audiometric pattern groups. Sixteen, or 53.4 per cent of these 30 audiograms were characterized by gradual and marked high tone loss and trough-shaped curves. Each of these patterns represents primarily perceptive type loss.

The etiological classification of heredity ranked next numerically, containing 14, or 9.3 per cent of the total number of cases included. These audiograms revealed either a moderate loss at all frequencies, or marked high tone loss.

In the birth injury group were found ten, or 6.6 per cent, of all the cases. The fact that eight, or 80 per cent of these ten, revealed a marked high tone loss, appeared significant. Fisher's Exact Significance Test was used to compare the birth injury etiology distribution with all other etiology distributions. This test yielded a probability of .00001, indicating that the observed distribution of birth injury etiology, according to pattern, could not have occurred as a result of chance factors alone; therefore, the marked high tone loss audiogram may be significantly related to hearing loss resulting from birth injury.

Nine, or 6 per cent, of the cases were found to have functional hearing loss, and were in the "psychological" etiologic group. Audiograms of these nine were rather randomly distributed among all patterns of hearing loss.

Seven, or 4.6 per cent, were included in the category "cause undetermined".

Rubella occurred in six, or 4 per cent of the cases analyzed. Although the numerical representation for this etiologic group does not lend itself to statistical analysis, it is interesting to note that two-thirds of the rubella cases show moderate loss for all tones or trough-shaped curve. This confirms the findings of Bordley and Hardy.[5]

The incidence of prematurity is small, with five, or 3.3 per cent of the total number of cases in this etiologic group. Two of these five had audiograms characterized by marked high tone loss, and thus in contour resemble the type of curve noted to be significantly related to case of birth injury.

Two of the ten etiologic classifications were represented by only one case each. These two were "drugs" and "head injury". The small incidence prevents any elaboration on the type of audiogram pattern each showed.

In summarizing the interpretation of the data, it can be pointed out that the present study of relationships between audiometric configurations and etiologies of hearing loss has lent support to the concept that causes of hearing impairment can be more definitively related to audiogram patterns. A number of interesting relationships have been noted. Although only a few of these were treated for statistical significance, it is believed that a larger sampling of the various etiologic groups would yield results that could be substantiated.

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FENESTRATION OPERATION IN THE POOR CANDIDATES.*

44 Cases Selected From 637 Operations.

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Tampa, Fla.

INTRODUCTION.

The primary purpose of this paper is not to advocate fenestration surgery in the poor candidate, but to find out what has been done with the fenestration operations in these cases. I have gone through the records of our 637 fenestration operations and selected the poor candidates for this analysis. There have been a total of 44 poor candidates, or 7 per cent (see Chart I), of the total series of 637. After analyzing these results, if there is adequate air-bone gap I am a little more inclined to advocate surgery in these poor candidates.

CHART I.

INCIDENCE OF OPERATED POOR CANDIDATES.

0 — 100	4
100 — 200	5
200 — 300	8
300 — 400	8
400 — 500	5
500 — 600	11
600 — 637	3
Total	44

Chart I. In the total series of 637 fenestration operations there has been a total of 44 operations upon poor candidates, or an incidence of seven per cent per one hundred fenestration operations.

In a previous paper I defined the poor candidate as the otosclerotic patient with a gradually sloping and converging air and bone conduction audiograph, with the bone conduction for the frequency of 2,000 below the 30 db level. For the purpose of this paper I have extended this definition to include a few

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selected cases in which I was in doubt about the benefit of fenestration surgery.

These cases of otosclerosis have been classified into two large headings: Associated Nerve Deafness, and Independent Nerve Deafness. The associated nerve deafness is that usually seen in otosclerosis and is characterized by the gradual loss of bone and air conduction for all the higher tone frequencies, producing a gradually sloping air and bone conduction audiogram. The term independent nerve deafness is used whenever there has been some other type of nerve deafness combined with otosclerosis, as a high tone nerve deafness, or endolymphatic hydrops. Under the cases of the associated nerve deafness there have been four cases of the so-called malignant otosclerosis, with rapid loss of hearing the greater for the high tones, and pink promontory of the cochlea. The classification of these cases is shown in Chart II.

CHART II

NERVE DEAFNESS IN OTOSCLEROSIS.

I	Associated Nerve Deafness	33
	1 A Malignant Otosclerosis	4
II	Independent Nerve Deafness	
	1. High Tone	4
	2 Hydrops	2
III	Osteogenesis Imperfecta	1
	Total	44

At the meeting of the Southern Section of the American Laryngological, Rhinological and Otological Society in 1954, I presented the results of the fenestration operation in the ideal candidate. It is easy enough to define the indications, and to advise these ideal candidates in which there is a greater than 90 per cent possibility of obtaining and maintaining a worthwhile hearing improvement through fenestration surgery. It is not so easy to evaluate and advise the poor candidates. These poor candidates must be individually selected, and only the surgeon himself can decide whether or not the patient will be happy with the partial result.

As previously stated, these candidates are usually advised as follows: "After a fenestration operation you will still need a hearing aid for group gatherings. I would encourage you to continue the use of the hearing aid and to forget about your fenestration operation; however, I cannot deny you the partial benefit which you might receive through fenestration surgery". These patients are further advised they would have a 60 to 70 per cent possibility of obtaining a worthwhile partial hearing improvement. They are advised to tell their friends that they will still need to use an aid after surgery. Regardless of the result, they are advised to continue to wear the hearing aid for the first six weeks after they return to work, then to discard the aid only under those circumstances in which they know that they can get along.

ASSOCIATED NERVE DEAFNESS.

The 33 cases of associated nerve deafness have been divided into two groups. The first group of 25 cases in which the bone conduction at 2,000 was above the 40 db level, and the second group of eight cases in which the bone conduction at 2,000 was below the 40 db level. A few of these cases were unable to hear the bone conduction at the frequency of 2,000. In compiling these results to give the average, we have included the two cases who received less than 10 db improvement.

AVERAGE IMPROVEMENT.

Fig. 1 shows the average improvement in the cases with 30 db air-bone gap, and Fig. 2 shows the average improvement in these cases with less than 30 db air-bone gap at the frequency of 2,000. This composite audiogram (see Fig. 1) shows the average result in the 25 cases with bone conduction between the 30 and 40 db level at 2,000. These cases showed an average improvement of 33 db for the low tones, 32 db for the speech frequencies, and an average of 8 db for the higher tone frequencies. In greater detail this improved the hearing from the 57 db hearing level to the 24 db level for the lower tone frequencies of 125 and 250. The hearing was improved from the 70 db level to the 38 db level for the speech frequencies of 500, 1,000, and 2,000. The hearing was improved from the 79 db level to the 71 db level for the higher tone frequencies of 4,000 and 8,000. At the most critical frequency of 2,000 these patients experienced a hearing improvement from the 73 db level to the 43 db level, or an average improvement of 30 db. This hearing improvement enables most of these patients to understand individual conversation. By and large they are grateful and happy patients. I have no hesitancy in recommending the fenestration operation in these cases of moderately severe otosclerosis in which there is a 30 db air-bone gap for the speech frequencies.

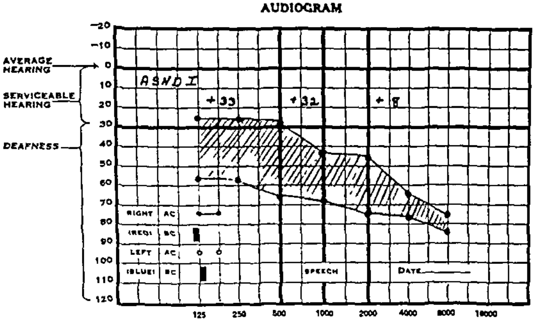

Fig. 1.

This composite audiogram shows the average result in the 25 cases of associated nerve deafness, with the pre-operative bone conduction between the 30-40 decibels level for the critical frequency of 2,000.

The second composite audiogram (see Fig. 2) shows the average improvement in those patients with associated nerve deafness in which the bone conduction was below the 40 db level for the critical frequency of 2,000, and in which the air-bone gap was less than 30 db for this critical frequency of 2,000. These patients showed an average gain of 26 db for the low tones, 29 db for the speech frequencies, and 6 db for the high tones. In more detail, the hearing was improved from the 50 db level to the 24 db level for the low tone frequencies of 125 and 250. The hearing was improved from the 66 db level to the 37 db level for the speech frequencies of 500, 1,000 and 2,000; the hearing was improved from the 92 db level to the 86 db level for the higher tone frequencies. Note on the figure, that the critical frequency of 2,000 shows the hearing was improved from the 78 db level to the 54 db level. With this sloping post-operative audiogram, these patients can hear about two-thirds of normal conversation. They must lip-read in order to understand the additional one-third of conversation. We advise all these patients to continue wearing the hearing aid, although with the concomitant loss at the frequency of 2,000 in the unoperated ear, most of them state that they can hear about as well in the operated ear as they can with the hearing aid in the unoperated ear.

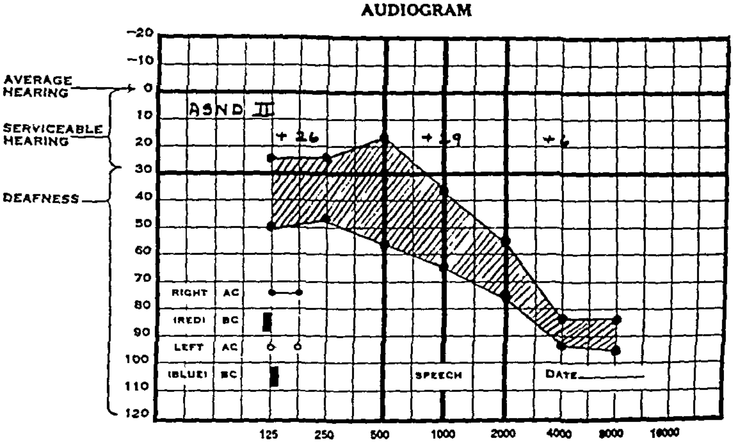

Fig. 2.
This composite audiogram shows the average improvement in those patients with associated nerve deafness where the pre-operative bone conduction was below the 40 decibels level for the critical frequency of 2,000. In Fig. 1 and Fig. 2, the composite average is based upon the best post-operative audiogram in the individual cases.

Included in the second group of associated nerve deafness are four cases of so-called malignant otosclerosis which deserve special consideration. By malignant otosclerosis we mean those cases of rapidly progressive hearing loss, with associated high-tone nerve deafness, and the pinkish promontory of the cochlea. One of these cases had about a 70 db hearing loss in both ears and was operated on in 1948. Three years post-operatively, she had maintained a 15 db hearing improvement in the operated ear, and had almost a complete loss of hearing in the unoperated ear. Today she can still wear the hearing aid in the operated ear and has almost complete loss of hearing in the unoperated ear. It is my firm opinion that the fenestration operation has preserved the hearing for many additional years in this 23-year-old female.

Another of these cases is one of the most severe cases I have ever operated upon. This was done at the patient's insistence, and with thorough understanding of the limited possibilities. This patient had a 95 db hearing loss for the low tones and no bone conduction at the frequency of 2,000. This patient experienced a 27 db hearing improvement for the lower tones of 125, 250, and 500, and a 15 db hearing improvement for 1,000 and 2,000. Although this gives this very hard-of-hearing patient little subjective hearing improvement, it may be that in the years to come the fenestration operation will have preserved some of his hearing. This is equivocal surgery, and I do not recommend the fenestration operation in such cases; however, if I had such a deafness I would certainly have a fenestration operation. Not included in these averages is a fifth patient with severe "malignant otosclerosis", who stated that he would commit suicide if we did not operate upon him. Finally, at his doctor's insistence, we proceeded with a fenestration, obtained the anticipated poor result, and the patient subsequently hung himself.

INDEPENDENT NERVE DEAFNESS.

The cases of independent nerve deafness are divided according to type. First, there have been three cases with an almost complete loss of high tones in the presence of an otherwise flat air conduction audiogram, and good bone conduction for the existing frequencies. These patients showed a 26 db hearing improvement for the low tones; 22 db hearing improvement for the speech frequencies, and a 5 db improvement for the higher tone frequencies. Second, we have operated on two cases with probable hydrops of the labyrinth. At the time of the operation the operated ear was the poorest ear. In both cases the operated ear is now the better ear. In one case the patient has good speech perception in the operated ear, there has been some decrease in the tinnitus in the operated ear. In the other case there was a 20 db hearing improvement, bringing the hearing in the operated ear to about the same level as that in the unoperated ear, but no worthwhile improvement so far as the patient was concerned. This is a disgruntled patient whom I have not seen in recent years. I have no conclusions to offer in those isolated cases where there is an associated endolymphatic hydrops in the presence of otosclerosis.

CLOSURES.

There have been no known closures in this group of 44 cases. In the last 300 cases there have been less than 2 per cent known closures. The fenestra have been made with sharp hollow ground margins under irrigation and under 7, 15, and now 40-power magnification.

COMMENTS.

The review of these poor candidates for fenestration surgery has revealed that the fenestration operation is of greater value than I had anticipated. In otosclerosis with the typical associated nerve deafness it is my opinion that the fenestration operation is a thoroughly worthwhile procedure, even though we can anticipate only a partial restoration of hearing. This opinion is substantiated by an average improvement of approximately 30 db for the speech frequencies in these cases of associated nerve deafness.

The patients with moderately severe or severe otosclerosis are very grateful for their partial hearing improvement. If the operation enables them to understand normal conversation at three to five feet, they are very pleased with their restoration of hearing.

The non-fenestrating otologists and the general public are inclined to be critical of the fenestration operation in these cases, because the patients have had the operation and they are still hard-of-hearing. The plea of this presentation is for tolerance. When one considers that these patients were profoundly deaf and completely out of auditory contact with the world, and that they have been improved to the ability to understand most individual conversations at the distance of a few feet, and that they can hear most ordinary noises, this seems to me to be very worthwhile surgery.

SUMMARY.

1. Forty-four fenestration operations on poor candidates have been selected from a total series of 637 fenestration operations and the results analyzed.

2. The poor candidates were subdivided into otosclerosis with typical associated nerve loss, and into otosclerosis with independent nerve deafness.

3. The cases of associated nerve deafness have obtained an average gain of about 30 db in the speech frequencies, improving their hearing from auditory isolation, to contact with the world.

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THE NEWCOMB AWARD.

At the Seventy-seventh Annual Meeting of the American Laryngological Association held at the Seignory Club, P. Q., Canada, May 13-14, 1956, the Newcomb Award of the American Laryngological Association was presented to Dr. Dean M. Lierle, of Iowa City, Iowa, "for his significant contributions to Laryngology and Rhinology and to the American Board of Otolaryngology."

THE ROLE OF THE CRICOTHYROID MUSCLE IN TENSION OF THE VOCAL CORDS.

An Experimental Study in Dogs Designed to Release Tension of the Vocal Cords in Bilateral Recurrent Laryngeal Nerve Paralysis.*

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That a way would be found to free the patient with bilateral recurrent laryngeal nerve paralysis from the lifetime use of a *tracheotomy tube has been in my mind and hope for many* years. This has been the ambition of others also, and many operations, such as the King, Kelly, and Woodman[1] operations, had the same purpose; however, they involved removal of the arytenoid cartilage on one side, changing the position of the vocal cord on that side, working in a difficult, deep corner in a more or less long operative procedure—or of moving one cord away from the median line. They presumed the acceptance in more than mere principle of the Semon[2,3] law, which states that "in bilateral recurrent laryngeal nerve paralysis adduction prevails longer than abduction because the posterior cricoarytenoid muscle, which controls abduction, fails before the cricoarytenoid lateralis, which controls adduction."

Murtagh and Campbell[4,5] lent support to this law by observation in animal experiments that the fibers of the adductor muscles were greater in size and number and more heavily medulated than those of the abductor muscles. General acceptance of this law led to the development of the King, Kelly, and Woodman[1] operations. Clerf and Baltzell[6] questioned the validity of the Law on the ground that the interarytenoid muscle is innervated by the external subdivision of the superior laryngeal nerve as well as by the recurrent laryngeal nerve; however, Lemere[7] showed that this branch of the superior laryn-

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geal traverses the muscle only to supply the underlying mucous membrane. Semon's observations were made largely in cases of injury to the nerves by tumors, aneurisms, and other conditions which produce gradual and varying degrees of imbalance in power between the abductor and adductor fibers; but the Law does not explain the paramedian position of the cords under all possible conditions and especially after complete surgical division of the nerves. It seemed obvious that it made little difference which muscle was stronger in its activity if both muscles failed in their activity at the same time.

During the years 1932-1934 I[13] had occasion to observe a fairly large number of recurrent laryngeal nerve paralyses following total thyroidectomy. These cases were done for the purpose of reducing the metabolic rate and lessening the work of the heart in chronic heart disease, in a study by Blumgart, Levine, and Berlin.[12] Most of the cases I observed that had a recurrent laryngeal nerve paralysis were unilateral and required no tracheotomy; however, the paramedian position of the cord *in all* cases was striking. The more *I* pondered on this the less convincing the Semon explanation seemed. I could well understand that, in a gradual pressure by tumor or aneurism on a recurrent nerve, paralysis of the muscles might occur in an uneven manner; but when both muscles were inactivated at the same time the resultant position of the cord should not favor either abduction or adduction.

In 1883 F. H. Hooper,[8] of the Massachusetts General Hospital, reported in "Experimental Researches on the Tension of the Vocal Bands" that the cricothyroid muscle was innervated by the external branch of the superior laryngeal nerve, fixed at the lower border of the thyroid cartilage and attached to the upper border of the cricoid cartilage. In contraction he observed that this muscle pulled up the cricoid cartilage to tense the vocal cord. He also explained the importance of the blast of air from below and the use of the extrinsic laryngeal muscles in causing higher degrees of tension, enabling the singer to reach higher notes. This was the first instance in which serious attention was paid to any factor other than the recurrent laryngeal nerves in relation to tension and the mobility of the vocal cords.

It seemed best to study this problem from a fresh point of view, leaving Semon's Law in abeyance for the time being; therefore I undertook a study of many postmortem larynges. It was quite evident that the vocal cords now were widely open and gave apparently sufficient space for respiration. This was really the cadaveric position. In contradistinction, in the living, when only the recurrent laryngeal nerves are bilaterally severed, the cords are in the paramedian closed position. This is definitely not the cadaveric position and implies that another nerve or muscle must be concerned in keeping these cords in tension and in the paramedian position. A further study brought realization of the fact that this tension was assisted by the pivotal action of the inferior cornus of the thyroid cartilage, attached by a synovial joint to the cricoid cartilage on each side, assisted in tensing the vocal cords by contracting the cricothyroid muscle in bringing up the cricoid cartilage toward the lower border of the thyroid cartilage, and this muscle, the cricothyroid, was innervated by the external branch of the superior laryngeal nerve.

Woodman[1] also seems to have realized this, because in his procedure he also released this attachment. A postmortem larynx, in contradistinction to a bilateral recurrent laryngeal nerve paralysis in the living, shows the vocal cords to be wide *open instead of closed. It seemed to me that it would be wise* to reconsider the innervation of all the intrinsic muscles and nerves of the larynx to determine all the factors concerned with the opening, closing, and tensing of the vocal cords.

MUSCLE.	NERVE.	ACTION ON VOCAL CORD
Cricoarytenoid Posterior.	Recurrent Laryngeal.	Abduction.
Cricoarytenoid Lateralis.	Recurrent Laryngeal.	Adduction.
Cricothyroid.	External Branch of the Superior Laryngeal.	Tension.
Arytenoideus.	External Branch of the Superior Laryngeal and partly by the Recurrent Laryngeal.	Tension.
Thyroarytenoid.	Recurrent Laryngeal. Rest of the Superior Laryngeal.	Abduction. Sensory.

easily manipulated in any direction. We tried the effect of merely severing the cricothyroid muscle and the inferior cornu on the same side. Although this succeeded it seemed that the complete work on both sides would carry greater assurance of success and nothing would be lost.

During this study my attention was called to an article by N. D. Fischer[10] entitled "Preliminary Report on the Application of the Motor Function of the Superior Larnygeal Nerve." In this paper Dr. Fischer suggested that the motor division of the superior laryngeal nerve in man has the same function as in the dog and that in dog experiments in which he had cut the right recurrent nerve and the right superior laryngeal nerve, the right vocal cord remained in the so-called cadaveric position and allowed for a good glottic chink. Since this procedure destroys laryngeal sensation, it would seem to be more desirable to cut the external branch of the superior laryngeal nerve alone, or to sever the cricothyroid muscle so as not to interfere with sensation. Moreover, as shown by Dilworth[11] and others, the free anastomoses between the branches of the superior and inferior laryngeal nerves would possibly make it difficult to find the external branch of the superior laryngeal nerve or to prevent complete loss of tension, which the anastomoses might defeat. For that reason we decided that it would be wiser to remove the cricothyroid muscle so as to attain as near complete loss of tension as possible. In all, fifteen dogs were used.

May I here describe a few of the procedures carried out on these dogs, and their purpose.

Experiment No. 1. Under general anesthesia, complete severance and removal of the cricothyroid muscle from the superior margin of the cricoid cartilage and from the lower border of the thyroid cartilage, and at the same time, severance of each inferior cornu from the thyroid cartilage and detachment from their attachments to the lateral surfaces of the cricoid cartilage. Ten days later examination of the larynx showed complete abduction but incomplete adduction, so that the vocal cords could not approximate evenly in the median line. This lent support to the presumption that tension was destroyed to the degree that the cords did not approximate at the median line.

Experiment No. 2. Only the left recurrent nerve was severed and the cricothyroid muscle removed on the right. Examination of the cords showed left recurrent paralysis, with the right overacting beyond the median line. One week later the right recurrent laryngeal was severed and the cricothyroid similarly treated as in the first operation. There seemed to be so much space at the end of the procedure that tracheotomy was not done—with the result that edema caused obstruction and no opportunity for continued respiratory activity—so that two days later the dog was found dead.

Experiment No. 3. The entire cricothyroid muscle was removed together with both inferior cornua of the thyroid cartilage and severance of the right recurrent nerve. Postoperatively, the right cord was immobile but was moved away from the median line, and the left cord was mobile but relaxed away from the median line. Tracheotomy was done. Ten days later the tracheotomy tube was extruded because it was blocked with secretion, but the tracheal opening was free and respiration was unimpeded. The tube was reinserted but removed after a few days because there was no edema of the cords and there was 5 mm. of space between them. This dog was observed for two months, and had no respiratory difficulty.

Experiment No. 4. The left recurrent laryngeal nerve was severed and the cricothyroid muscle removed, together with both inferior cornua of the thyroid cartilage. Postoperative examination showed left paralysis. No tracheotomy was done. One week later the right recurrent nerve was severed, but no tracheotomy was done, because there was no evident edema from the previous operation. This dog was observed for a number of months and no respiratory difficulty was noted.

Experiment No. 5. Both recurrents were severed and tracheotomy tube placed. Both cords were in the paramedian position. Ten days later the cricothyroid muscle was removed and the inferior cornua of the thyroid cartilage, also. The tracheotomy tube was left in place. One week later there was 6 mm. of space between the cords. The tracheotomy tube was removed, and there was no respiratory difficulty.

Experiment No. 6. Both recurrents were severed and only the left half of the cricothyroid severed, and dog was tracheotomized. Both cords were immobile, but there was 4 mm. of space between them. The tracheotomy tube was removed one week later, and respiration was unimpeded.

Summary: On the principle that abduction, adduction, and tension are the three main factors concerned with the motility of the vocal cords and free respiration, a series of experiments was carried out on dogs, first, to sever both recurrent laryngeal nerves and thus insure bilateral recurrent laryngeal paralysis. Tracheotomy was then carried out. Later, the cricothyroid muscles were detached from the lower border of the thyroid cartilage and the inferior cornua of the thyroid cartilage severed from their attachments to the lateral surfaces of the cricoid cartilage. When, by observation, it was evident that the glottic chink was wide enough for normal respiration, the tracheotomy tube was removed and the wound closed. The dogs were able to breathe freely without apparent discomfort thereafter. It is hoped that this method may be of use in human beings, and make unnecessary more complex procedures involving removal or displacement of the arytenoid and sewing back of the vocal cords. The procedure herein described is so simple that it would seem to merit trial in human beings.

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475 Commonwealth Avenue.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Home Study Courses.

The 1956-1957 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, which are offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Detailed information and application forms can be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minnesota. Registrations should be completed before August 15.

HEMICRANIA—OR ONE-SIDED SPHENOPALATINE ETHMOID HEADACHE.*

WM. H. TURNLEY, M.D.,
Ocala, Fla.

The Greek word "hemicrania" means half-head, and the term was first adopted by Galen (201-137 B. C.) but, in the course of time, underwent considerable etymological ill-treatment, until it was corrupted from "hemicrania" to "emigrana", then "migrana", and finally to the French term "migraine". I am not going to discuss the other side, for this headache is predominantly on one side; although, after a long attack, it may become a generalized headache on both sides, or the patient just does not care, and says it is all over the head. The origin seems to be definitely located in the sphenopalatine ganglion region of the nose, between the posterior ethmoid cells and nasal septum. The syndrome may radiate from this spot to the entire side of the body in extreme cases, simulating the whole gamut of headaches from tic to epilepsy.

One reason I chose this subject at this time is because the literature has gone overboard with the emphasis on "vascular headache", but I still think a focus of infection plays an important role, whether it be bacteria, virus or their mutations; and I might say in the beginning, I have no use for foci of infection anywhere, at any time, and that goes for germs in the human body, weeds in the garden, and crooked politicians in government.

Another reason I chose this subject may be a local one, for I practiced otorhinolaryngology in New York City and Connecticut for 25 years, then suddenly moved to Ocala, Fla. In the past four years, I have seen more of this type of headache here in this part of the South than I did in a metropolitan northern area in ten years. I believe it is due to the predominance of the Anglo-Saxon or Caucasian people in this region,

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who have a high, narrow nose with pressed-in wings, in contrast to the mixture of races of people who have a broader nose.

In reviewing the literature on this subject over the past decade, I find there are very few articles concerning the sphenopalatine syndrome, but so many volumes have been written on histaminic cephalalgia, endocrine headache, trigeminal neuralgia, tension headaches, myalgia, allergic headaches, vascular phenomena, and atypical everything, that it is, at least, confusing. I have written this paper from my own practice and experience, and if some of you disagree with it, that's good. We all disagree on some things, and the Bible says there will never be a time when any two men are of the same accord.

A review of the anatomy of the sphenopalatine area is adequately described in Morris' *Anatomy*. The nasal fossae are two irregular cavities situated on each side of the median vertical septum. Each fossa communicates with the sinuses of the frontal, sphenoid, maxilla, and ethmoid bones.

The roof is horizontal in the middle but slopes downward in front and behind. The anterior slope is formed by the posterior surface of the nasal bone and the nasal process of the frontal; the horizontal portion corresponds to the cribriform plate of the ethmoid and the sphenoidal concha; the posterior slope is formed by the inferior surface of the body of the sphenoid, the ala of the vomer, and a small portion of the sphenoidal process of the palate. The sphenoidal sinus opens at the upper and back part of the roof into the spheno-ethmoidal recess, above the superior meatus.

The septum is the median wall which is usually deflected from the middle line to one side or the other and occasionally has large spurs. It also may contain ethmoid cells superiorly. The lateral wall is the most extensive and the most complicated on account of the formation of the meatuses of the nose. The superior meatus, the shortest of the three, is situated between the superior and middle nasal conchae, and into it open the orifice of the posterior ethmoidal cells and the sphenopalatine foramen.

The nerve center is situated in the sphenopalatine, or Meckel's, ganglion, and is associated with the maxillary nerve. The ganglion is a reddish-grey body and measures about 5 mm. It lies deeply in the pterygopalatine (spheno-maxillary) fossa at the lateral side of the sphenopalatine foramen and in front of the anterior end of the pterygoid (Vidian) canal. It is attached to the maxillary nerve, from which it receives its sensory root, and it is connected with the Vidian nerves, which furnish it with motor and sympathetic filaments.

The Vidian nerve is formed by the union of the great superficial and deep petrosal nerves in the foramen lacerum to the sphenopalatine ganglion. The Vidian nerve is joined by a sphenoidal filament from the otic ganglion, and it gives branches to the upper and back part of the roof and septum of the nose and to the lower end of the Eustachian tube.

The anatomical and clinical evaluation of head pain attributed to the sphenopalatine ganglion was very carefully studied by Greenfield Sluder in 1908. He thought that the origin of this type of headache is by extension from the posterior ethmoid and sphenoid sinuses and said, "In no other part of the body is a sympathetic ganglion or sensory ganglion so exposed to surface influences." Of course, headache may have more than one cause, but, in this particular type there is swelling and infection whether it be of bacterial or virus origin, or both. The pathologic basis may be in dispute, but most students believe that a disturbance in the cerebral vasomotor mechanism underlies all cases. This may be just another symptom of the original sphenopalatine disturbance, for wherever there is swelling, from infection or not, there is pressure on the nerve end organs, which is transmitted as pain.

Sluder also thought that the sphenopalatine ganglion is not separated from the nasal cavity by bone; therefore, many anatomists are not willing to accept this as the absolute interpretation, and that leaves the anatomic solution of this referred pain problem still unsettled. Rather than get into this controversy, I refer you to articles by Robert B. Lewy, in the *Journal of Laryngology and Otology,* Vol. 65, No. 1, Jan., 1951; Dr. David R. Higbee, *Transactions of the American Academy of Ophthalmology and Otolaryngology,* 52: 283, March, 1948.

I am merely trying to tell you the clinical results I have obtained from a careful and thorough submucous resection, especially of the posterior and superior nasal septum in this particular type of headache.

The first sign of this neuralgic syndrome may start as a stuffy feeling in the nose, with all the symptoms of a cold. The attack may come on rather suddenly, following a change of atmospheric pressure, temperature and humidity. This dull feeling in the nose is always on one side at first, the eye aches deep in the back, and the patient may think he needs glasses or a change of lenses. The pain may be across the bridge of the nose or a tenderness of the upper inner angle of the orbit of the eye, or sharp pain in the temple. There is often an area on top of the head which is sensitive even to touch. Many cases have a definite tender spot just back of the mastoid process, as Sluder describes it, 5 cm. post to mastoid tip. The ache is always in the back of the head and occiput, neck and shoulder and down the deltoid muscle of the arm to the elbow, or even to the fingertips.

Some describe a low-grade ache over the maxilla, and along the Eustachian tube; but the most common course is around the eye, cheek, temple, top of head, occiput, back of neck and shoulder. Sometimes, this syndrome will last for a few days and clear up voluntarily under certain conditions. It may last for several years at a time, as a constant, low-grade ache; again, there may be excruciating attacks, and the patient may become almost hysterical, with nausea and vomiting. These patients are sure they have a brain tumor, and may contemplate suicide. I believe trigeminal neuralgia, especially of the ophthalmic and maxillary branch, is just a more drastic form or different manifestation, of this same syndrome; likewise, herpes zoster ophthalmicus and oticus are of the same origin.

After taking a further history of these cases, there is generally a story of always having a cold, and eventually the patients may tell you that they have lost their sense of smell (anosmia). Then you know they have a definite ethmoid sinus infection of many years' duration. A post-nasal drip is nearly always a forerunner, and it may not necessarily be pus; later, there may be a bloody discharge.

On examining the nose, the casual observer may say the patient has no crooked septum or sinus condition; but, after shrinking the turbinates, a closer examination, far back and high up in the post-ethmoid region, will show you the septum and turbinates are stuck together so tightly that a probe can not be passed between them. The septum may be swollen several times thicker than normal, but as soon as you separate it from the superior turbinate with a probe, tipped with cotton soaked in 10 per cent cocain, the patient says, "Oh! I can breathe now; my whole head feels clearer, and that ache in the back of my neck is gone."

Here again in this region, that old adage "Symptoms are more important than signs" holds true, for an X-ray film seldom shows anything unless there is a definite bacterial infection in the posterior ethmoid cells. The sphenoid sinus does not seem to be involved as often as was once thought. I believe transillumination gives just as much information as an X-ray does in these cases, and it can be done more quickly. Seldom is there any temperature, except in an acute fulminating condition.

There is quite often a Eustachian tubal catarrh, with a slight loss of hearing for the low 128 fork, bone conduction greater than air conduction. Weber test lateralizes to the affected side.

Children do not seem to have this type of headache so much as adults. This is probably because they are more flexible, not so sensitive to pain as adults, don't know how to describe their condition, or that the growing process has not developed the septum to the final stage causing pressure; but when the headache is present, it should be corrected by a submucous resection at whatever age they are. I have often wondered whether a child delivered by forceps is more likely to have a depressed bridge of the nose, thereby deflecting the septum, than those delivered without instrumentation. Patients with a high acute-angle, bony palatine arch, nearly always have a deflected septum.

Trauma late in life, from a blow that may break the bone or distort the septum, does not seem to be an etiological factor for this headache. I believe the cause is more of a developmental origin. A thermostatic imbalance, along with a change of barometric pressure and humidity, may be an exciting physical factor, while a disturbed endocrine balance may be an exciting physiological factor. In other words, anything which causes the turbinates to impinge against the septum permanently and block off the ethmoid air drainage, may start the syndrome. Males and females are about evenly divided as to sex origin.

There seems to be two schools of otolaryngologists: those with a medical outlook, and those with a surgical outlook. We need both, for there is still plenty of surgery to do, and a whole new world of therapy to come. Some day we will find out what a cold or sinusitis really is, but, for the present, I think the first running nose a baby has, at the age of a few months, is his first cold or ethmoid sinusitis, and from then on it may progress, or subside, depending upon his natural resistance and the various factors that influence him. The cold may be a virus that just gets the soil ready for the seed, so to speak. Then the secondary invaders of bacteria gain entrance, and one has a real bacterial sinusitis; but all this is a vicious cycle, and as the baby grows, so does the septum of his nose, and if this septum does not have room to expand, it will bend to one side or the other, and block off the ostia of the ethmoid sinus. The nose must have free circulation of air all the time, just like the rooms in a house, for if there isn't good air circulation, things get stale and mouldy. The ostea of the posterior ethmoid cells are small, and it does not take much swelling of the mucosa, or pressure from a curved septal wall, to block them.

Like the dirt in the eye of a needle, it does not take much dirt to block the eye, but until you get this little amount out, the needle is useless for sewing, regardless of the other dirt around the needle. When you look in a nose and see an obvious large deviated septum or spur on the floor, this is not nearly so bad as that gentle little compensatory curve on the septum, situated far back, and at the top of the septum. You can hardly see this curve, especially if the turbinates are swollen; but, if you shrink the turbinates with a solution of cocain, then you can see this deflection, or obstruction. As I said before, a little blockage in this area does more harm than the big, obvious deviation below. The patient seems to get plenty of air under the inferior and mid-turbinate, but yet he says he has a feeling of stuffiness up high, and he may complain of a musty smell. This is one of the most sensitive areas in the body, and if you don't believe it, just try to probe it on yourself. It is amazing how excruciatingly painful just the slightest pressure can be. That is why so many doctors do not get this area separated, for the patient won't let him place the probe there; but it is imperative that you cocainize this area, so wait for a minute or two until the cocain takes effect, and then place a fresh probe, with a little 10 per cent cocain, further up. When you hear a clicking sound, like that of two glued pieces of paper pulled apart, then you know you have let air into this area, and the patient gives a sigh of relief and says, "Why this is the best I have breathed through my nose in months." Almost immediately, the pain on the top of his head, or behind his ear, or back of his neck, is relieved. It is very spectacular, and this is a good sign you have hit the spot.

Sometimes just one treatment is sufficient to relieve a patient for several months, or you may have to do this every day or so for several times. He can be given a mild spray, of chloretone inhalant, or your own pet Rx, but the best way to give this patient permanent relief, is to do a thorough submucous resection of the septum.

It is not necessary to describe this operation, except to say you must get that little curve up high and back in the ethmoid region, and to do that it is necessary to resect the septum below and front. Many beginners do a partial submucous resection, get a perforation, and stop, or think they have done a complete submucous resection. Not so; you might just as well have done nothing so far as relieving this type of headache. It is not a difficult operation to do, but it is a tricky one; like a game of golf, you must be accurate and complete the stroke all the way through to shoot par.

When a patient tells you he has had a submucous resection operation, don't believe him; look for yourself, even if it has been done by the best surgeon in "Podunk", or even if it has been done twice. There is one thing that seems to be avoided in the literature on surgery, and that is the criticism of poor tle blockage in this area does more harm than the big, obvious deviation below. The patient seems to get plenty of air under the inferior and mid-turbinate, but yet he says he has a feeling of stuffiness up high, and he may complain of a musty smell. This is one of the most sensitive areas in the body, and if you don't believe it, just try to probe it on yourself. It is amazing how excruciatingly painful just the slightest pressure can be. That is why so many doctors do not get this area separated, for the patient won't let him place the probe there; but it is imperative that you cocainize this area, so wait for a minute or two until the cocain takes effect, and then place a fresh probe, with a little 10 per cent cocain, further up. When you hear a clicking sound, like that of two glued pieces of paper pulled apart, then you know you have let air into this area, and the patient gives a sigh of relief and says, "Why this is the best I have breathed through my nose in months." Almost immediately, the pain on the top of his head, or behind his ear, or back of his neck, is relieved. It is very spectacular, and this is a good sign you have hit the spot.

a lot of factors that come into play. When the swamp dries up, the turtles, mosquitoes, and water lilies die, so when the ethmoid sinus gets aerated and drained, the streptococci and other germs diminish too. I believe it was Hippocrates, years ago, who advocated good drainage and aeration, wherever there is blockage to infection.

Sometimes the patient may have a definite chronically infected ethmoid condition. If so, one has to use his judgment as to whether to take off the superior turbinate, or do a partial ethmoidectomy. I have a great deal more respect for the ethmoid cells than I did as an intern. Now I leave them alone, but if they are definitely destroyed and cystic, it may be necessary to remove or open them; for an infected hidden ethmoid cell may suffice to keep in action the entire toxic syndrome, if it cannot have drainage.

A submucous resection is easier to do under local anesthesia, rather than under general anesthesia, but children and some adults require a general anesthesia. I believe this operation should be done early in life rather than late, as formerly advocated. No harm can be done after 10 years of age; also, I think all asthmatic patients should have this operation done, whether they have headache or not.

As for the medical treatment of this type of headache, I am sure you are familiar with the best of modern drugs. Naturally, we want to give these people relief, but no drug seems to work too well, not even demerol or morphine, but cocainization of the sphenopalatine ganglion area will relieve most patients of pain for an hour, if not for a longer period of time. All of my extreme cases have had the "pharmacopoeia thrown at them"—histamine de-sensitization, anti-histamine, estrogens, nicotinic acid, vitamins, diets, ergotamine tartrate, amphetamine, cortone, and the vasodilators. Probably as good as any is a cafergot suppository, a dark quiet room and a warm bed. A drink of bourbon is better than a cigarette any time. The best prophylactic I know is to avoid getting chilled, or a cold, or an emotional upset.

I must mention virus as a type of infection in this area to be treated, since recent research has already produced an astonishing array of hitherto unknown viruses. At the last offia lot of factors that come into play. When the swamp dries up, the turtles, mosquitoes, and water lilies die, so when the ethmoid sinus gets aerated and drained, the streptococci and other germs diminish too. I believe it was Hippocrates, years ago, who advocated good drainage and aeration, wherever there is blockage to infection.

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THE MANAGEMENT OF FACIAL PARALYSIS ASSOCIATED WITH FRACTURES OF THE TEMPORAL BONE.*

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Definitive surgical treatment of facial paralysis due to basilar skull fracture is necessary in certain instances; and in these few properly selected cases, it may be accomplished with gratifying results.

Facial paralysis associated with acute head trauma indicates temporal bone fracture in nearly every instance. It is usually accompanied by bleeding from the ear on the involved side and rather rarely by cerebrospinal otorrhea.

The incidence of facial paralysis produced by acute head injury is somewhat difficult to determine, but a review of reported series of cases would indicate that it is relatively high if one considers only the middle and posterior fossa fractures. Grove[1] reported 29 cases of facial paralysis in his series of 211 skull fractures (14 per cent). In his exhaustive review of the literature pertaining to fractures of the petrous bone, he cited the statistical studies of many authors. He said: "According to the statistics of Siebenmann, Brunner, Voss, and

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Ulrich, transverse fractures cause a facial paralysis in about 50 per cent of the cases, and in the longitudinal variety in from 10 per cent to 18 per cent." A much higher incidence was recorded by Davis[2] who reported peripheral facial paralysis in 46 per cent of middle fossa fractures.

Even in large series of unselected cases of acute head injury, the incidence of facial paralysis is considerable. Turner[3] reported 46 instances in his series of 1550 acute head injuries of all types. With the exception of the olfactory nerve which was injured in 119 instances, it was the cranial nerve most commonly involved. In a series of 600 cases of head injury, Russell[4] found that facial paralysis occurred 17 times. It was *interesting to note that this represented about one-fifth of the* cases in which there had been bleeding from the external ear.

The most common type of skull fracture which may involve the facial nerve is one in the floor of the middle fossa. Rowbotham[5] states that in his experience, these are more apt to extend from the vault to the base than to be confined to the base alone. They may start in the temporal squama or the parietal bone and extend through the mastoid into the middle ear. Rowbotham states that the facial nerve is usually injured distal to the geniculate ganglion. Ulrich[6] stated that when the facial nerve is injured, the injury is in the region of the geniculate ganglion. Because of the strong buttress effect of the petrous pyramids, fracture lines extending from the vault are apt to be deflected horizontally along the anterior border of the petrous. Such horizontal fractures occur much more frequently than transverse fractures; thus the middle ear is involved much more commonly than is the otic capsule. The transverse fractures are primarily posterior fossa fractures which have resulted from terrific force applied through the condyles.

Kettel[7] describes two types of transverse fractures: the internal which involves the internal auditory canal and the cochlea; and the external which passes through the cochlea, vestibule, and Fallopian canal. In these, there is no tearing of the tympanic membrane and hence no bleeding from the external auditory canal, although there may be a hemotympanum.

Gurdjian[8] describes cases of combined transverse and longitudinal fractures that have come under his observation. In such fractures, there may be wide variations in the neurological signs, depending upon the position and extent of the fracture lines.

Ramadier and Caussé[9] describe an unusual type of fracture confined to the mastoid process which could involve the external canal and middle ear, injuring the facial nerve in its vertical course.

Facial paralysis complicating fractures of the temporal bone may be immediate or delayed. The immediate paralyses obviously denote a direct injury at the time of fracture which may represent laceration, stretching, or compression of the nerve by bone fragments. The delayed cases may be due to edema and hemorrhage, or, still later and less commonly, to infection.

There is an abundant literature on all aspects of skull fractures. Even a cursory review of articles dealing with fractures of the temporal bones is beyond the scope of this presentation. For the present purpose it has seemed sufficient to cite authoritative descriptions of the most common types of temporal bone fractures, and to note accepted opinions regarding the frequency of associated paralysis and the most common sites of injury to the nerve.

More important, perhaps, in consideration of the cases to be presented at this time, is a brief discussion of the prognosis of facial paralysis produced by a fracture through the temporal bone.

Kahn[10] and Gurdjian[8] agree that about 90 per cent of these cases recover completely, or at least quite adequately. Perhaps three or four of the remaining 10 per cent suffer complete and permanent paralysis, while the rest have poor or inadequate return of function. Kettel,[7] after an exhaustive study of the subject, concluded that of the immediate paralyses, 75 per cent of patients recovered completely; 15 per cent showed partial recovery, and in 10 per cent massive paralysis remained.

In Grove's[1] series of cases, there were 29 instances of facial paralysis of which five were due to transverse fractures, or at least fractures which extended through the labyrinth, and 21 due to longitudinal fractures. In one transverse and in five longitudinal fractures, the paralysis was complete and permanent. The others "either improved or cleared up completely."

Rowbotham[5] states that at least 90 per cent of cases recover. In his experience, only one case in 30 failed to recover, and that was in an individual who had a fracture through the internal auditory canal. He states that the "favorable cases show signs of improvement within two months and usually within two or three weeks." Rowbotham[5] declares further that "an incomplete injury of the facial nerve may later lead to facial spasm, a condition equally as disabling, and probably more disfiguring than facial paralysis." He concludes that although theoretically, routine decompression should be done in all cases, particularly the delayed ones, early operative interference is contraindicatd because of the high percentage of good spontaneous recoveries; however, he does advocate exploration of the nerve regardless of electrical reactions, after three months, in those patients who have shown no improvement.

Russell[4] expects spontaneous recovery of all forms of facial paralysis after injury, and does not mention decompression or other surgical procedures to alleviate the condition in his chapter in Brock's text.

Spurling[11] states that the prognosis for complete recovery of immediate cranial nerve palsies, due to skull fracture, is poor.

Browder[12] states that "except in rare instances when facial paralysis results from direct trauma to the facial nerve, surgical intervention is contraindicated. Satisfactory recovery may be expected to follow massage and electrical stimulation of the paralyzed muscles."

Turner[13] studied 70 consecutive cases of traumatic facial palsy of which 36 were immediate and 34 were delayed from two to eight days after injury. Of the latter group, 22 were partial and 11 were complete. In the delayed cases one patient, who developed an acute otitis media on the fifth day, failed to recover any function of the facial muscles, and two had incomplete recovery with mass motion after six and eight months. Of the 36 immediate cases, 19 complete and 17 partial, six had incomplete recovery which started at three months, and three remained completely paralyzed.

If the prognosis of facial paralysis due to skull fracture is good for spontaneous recovery in 90 per cent of cases, and this seems rather high after reviewing the literature on the subject, there is still that 10 per cent of such patients in whom surgical intervention is indicated when the lesion is surgically accessible. By careful assessment of the individual cases, the otologist should be able to select with discrimination those patients in whom exploration of the Fallopian canal is required.

The selection of patients for such a procedure demands careful attention to the details of history and examination. One must ascertain whether or not there was bleeding from the external ear at the time of the accident and if the paralysis was immediate or delayed, for nearly all patients who experience a delayed paralysis proceed to a satisfactory spontaneous recovery. Since specific information regarding the time of onset of the paralysis is too frequently omitted in the history of referred patients, it is felt that, generally speaking, a three months' waiting period is advisable before consideration of surgical intervention. Any physiologic block or neuropraxia should have recovered by that time. In most reported series of cases complete or satisfactory spontaneous recovery, if it is going to occur, is manifest in four to eight weeks. If, however, a response to Faradic stimulation which was previously absent has become demonstrable at the end of three months, further delay may be indicated but is probably inadvisable, for in such patients the degree of spontaneous return of function is likely to be unsatisfactory. The waiting period of three months does not militate against a good result from decompression, nerve suture, or nerve graft if such should be found necessary.

Examination should include careful otoscopic observation, Roentgenograms including Stenver's views, tuning fork and audiometric hearing tests, vestibular tests, percutaneous Faradic stimulation, and tests for lacrimation, taste, and salivation. In late cases, muscle responses to galvanic stimulation must be determined also.

Transverse fractures through the labyrinth resulting in facial paralysis with complete loss of cochlear and vestibular function usually carry a bad prognosis, for the site of nerve injury is likely to be inaccessible for repair in continuity. In these instances, substitution anastomoses with the hypoglossal or spinal accessory nerves may be required. Occasionally, however, repair in continuity in such a case may be possible if the transverse fracture is of the external type as described by Kettel.[7] An example of this type is described in Case 3.

Roentgenograms will usually demonstrate well a transverse fracture, but a longitudinal one may be more difficult to detect. Oblique films of the temporal bones are most apt to show those fracture lines extending from the vault into the middle fossa.

If cochlear and vestibular functions are preserved in a patient with facial paralysis due to temporal bone fracture, the facial nerve lesion is probably in a surgically accessible region.

The most valuable single test of facial nerve function has been Faradic stimulation. If good response to Faradism can be obtained, even if the use of 40 volts is required, the prognosis for at least some degree of spontaneous recovery is good. If the Faradic response is absent, it does not indicate the severity of the lesion. I agree with Kettel[7] who states that "a positive response may be a good prognostic sign, but nothing final can be deduced from a negative reaction." Electromyographic studies are valuable, but have not proved to be infallible. They have been of value during the period of waiting, for responses may be obtained prior to those induced by Faradism.

When, after three months, or better at an earlier date if the patient's general condition is good, if the paralysis has remained complete and there is complete reaction of degeneration, surgical exploration must be considered. At this time the localization of the lesion must be determined as accurately as possible. Tests for chorda tympani and stapedius nerve functions are valuable. For the stapedial nerve test, the binaural stethoscope is used as described by Tschiassny.[14]

Since taste testing for chorda tympani function has been unsatisfactory in many instances, we have been using in addition a technique for measuring the salivary secretion of the submaxillary glands. In this procedure, a No. 60 polyethelene catheter is placed in Wharton's duct on each side, The patient is asked to suck on a lemon for five minutes, during which time the output of saliva is measured. In cases of absent chorda tympani function we have found, as would be expected, a marked decrease in the amount of saliva secreted on the involved side. One reason which may account for some unreliability of testing taste on the anterior two-thirds of the tongue, to determine the function of the chorda tympani nerve, is explained by Crosby.[15] She states that in embryological development some fibers of the glossopharyngeal nerve may be drawn forward in the tongue and serve to conduct gustatory impulses in the absence of chorda tympani innervation.

When surgical exploration with anticipated nerve repair by decompression, end-to-end suture, or grafting is contemplated, the most important determination will be in regard to the function of the lacrimal gland. The Schermer test is very satisfactory, but much information can be gained from observation of lacrimation after irritating the nasal mucous membrane, either chemically or by means of a cotton tipped applicator. If there is a dry eye, and particularly if this is associated with loss of cochlear and vestibular function, exploration will probably be fruitless and nerve repair impossible, except by means of substitution anastomosis. Normal tearing indicates a lesion distal to the geniculate ganglion which should be accessible and amenable to surgical repair in continuity.

Usually a radical mastoidectomy is required, but in some instances simple mastoidectomy may permit of adequate exposure (see Case 1). This, of course, may be anticipated if it can be shown that the lesion is distal to the branch of the facial nerve supplying the stapedius muscle. In some cases, a modified radical mastoidectomy will be required. Every effort should be made to preserve the patient's residual hearing.

After such careful evaluation, one can proceed with some confidence in those patients in whom surgical intervention is deemed advisable.

Examples of facial paralysis demonstrating different types of nerve injury produced by temporal bone fractures will be presented in three case reports. The surgical technique used in each case was different from the others, and depended upon the pathological condition present.

Case 1: D. LeV., No. 811177, a white male, aged 22, was admitted to University Hospital on May 18, 1955, because of a complete right peripheral facial paralysis. On February 24, 1955, he had sustained a severe head injury with a linear fracture in the right temporal region from an automobile accident. It was reported that the facial paralysis had been immediate and that there had been bleeding from his ear at the time.

Routine objective examination of the pharynx, nose, and left ear revealed no significant pathological changes. The right tympanic membrane was intact although somewhat retracted, and the external auditory canal appeared to be normal. An audiogram showed hearing that was slightly depressed but nearly normal. The vestibular function was normal on caloric stimulation. He had a complete right peripheral type of facial paralysis with complete reaction of degeneration. No response was obtained on percutaneous stimulation with Faradic current of 40 volts. Other than the facial paralysis there was no neurological deficit. The patient had retained sense of taste on the anterior two-thirds of his tongue.

Roentgenograms revealed a fracture line in the right temporal region, which extended downward into the mastoid. The mastoid process appeared to be normally aerated (see Fig. 1).

Because of the continued complete peripheral facial paralysis which apparently had its onset at the time of the injury, the complete reaction of degeneration and absence of injury to the otic capsule, indicating that the traumatic lesion to the facial nerve was accessible, surgical exploration with an effort to effect nerve repair was advised.

On May 20, 1955, approximately three months after injury, a right mastoidectomy was performed. There was no evidence of any infection in the large pneumatic mastoid process. The fracture line was clearly visible as it extended over the mastoid cortex to the upper portion of the external auditory canal. In enlarging the aditus ad antrum, it was noted that the fracture had extended into the fossa incudis. After removal of the mastoid tip, the facial nerve was exposed throughout its vertical course and appeared to be normal up to the pyramidal segment. Further exploration in the region of the fossa incudis revealed that the short crus of the incus had been dislodged and that there were small spicules of bone in the region of the fossa, which were compressing the facial nerve. After removing these spicules the exposed nerve in this region appeared to be compressed to about one-third its normal size (see Fig. 2).

A little further rotation of the incus without disarticulating it, per-

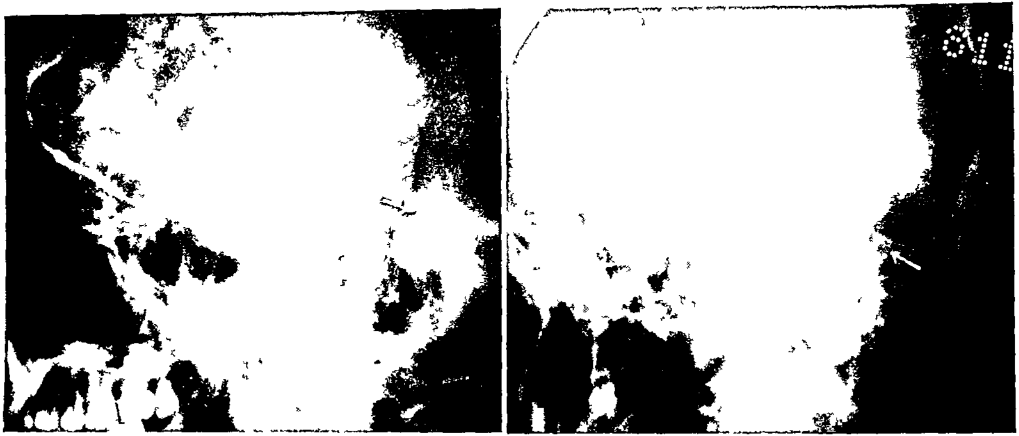


Fig. 1 Case 1 Roentgenograms demonstrating the fracture line in the temporal squama extending into the mastoid process indicated by arrows

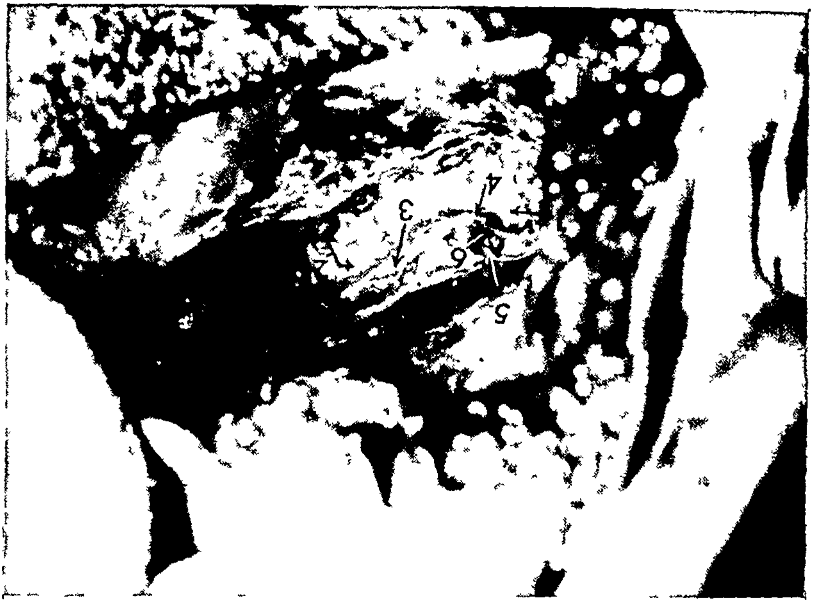


Fig 2 Case 1 Photograph taken during operation after completion of mastoidectomy and exposure of facial nerve

Arrows indicate 1 Horizontal semicircular canal, 2 Mastoid tip, 3 Vertical course of facial nerve, 4 Compressed nerve at pyramidal segment, 5 Incus, 6 Elevated short crux of incus

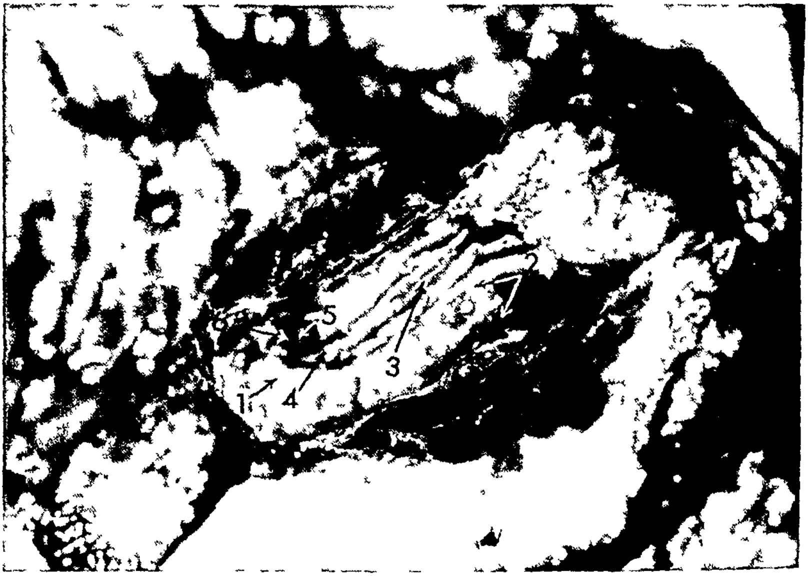


Fig 3 Case 1 Photograph taken at end of operation showing bulging of facial nerve after slitting the sheath

Arrows indicate 1 Horizontal semicircular canal, 2 Mastoid tip, 3 Vertical course of facial nerve with sheath opened, 4 Bulging pyramidal segment of facial nerve, 5 Incus, 6 Middle ear cavity mitted sufficient exposure to uncap the facial nerve in its entire intratympanic course and to examine the ossicular chain, which was found to be intact The nerve had an essentially normal appearance except in its pyramidal segment Under magnification the sheath of the nerve was slit from the stylomastoid foramen upward throughout its intratympanic course After [illegible]

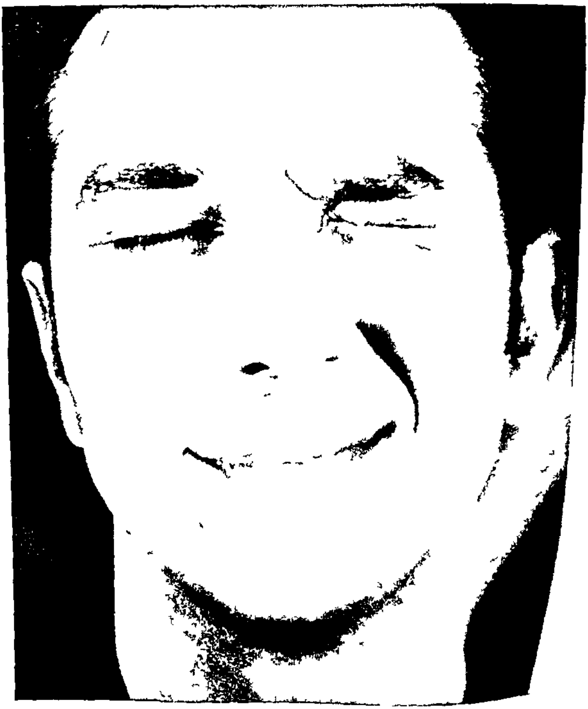


Fig 4 Case 1 Photo[illegible]

normal appearing size in the [illegible] Fig 3) No evidence of [illegible]

The patient's convalescence [illegible] the hospital on the fifth [illegible] of motion was noted in his [illegible]

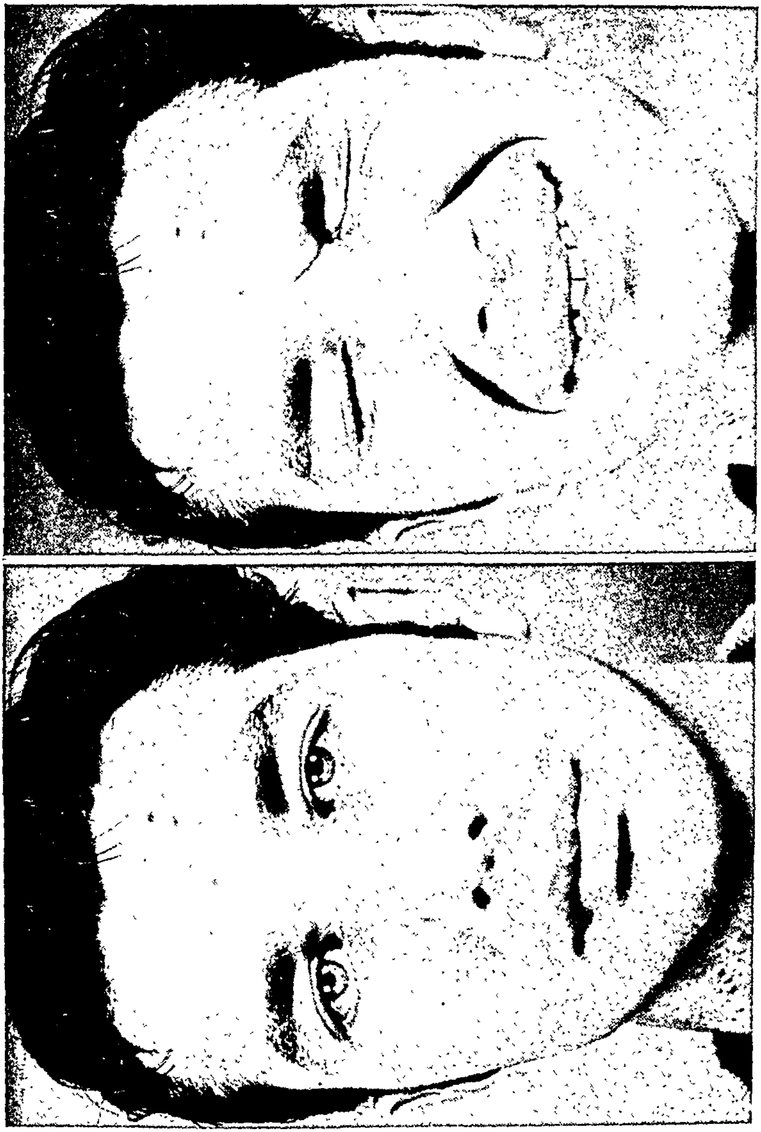


Fig. 5 A.

Fig. 5 B.

Fig. 5. Case 1. Photograph of patient taken 3 months after operation showing further improvement.

By June 22, 1955 (one month postoperative) the patient showed considerable return of function. He could close his eye tightly but did not have as much power on the operated side of the face as he did on the normal side (see Fig. 4). There had been preservation of the preoperative hearing.

He was last examined on August 8, 1955, at which time his general condition was excellent. Functional return of facial muscles was adequate but not complete, in that there was weakness of the lower lip, weak frontalis action, and a little mass motion (see Fig. 5). The audiogram showed nearly normal hearing as seen in Fig. 6.

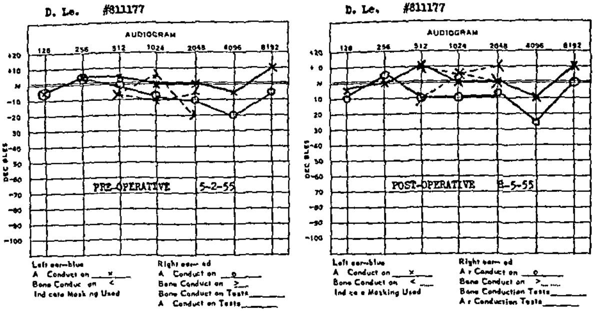


Fig. 6. Case 1. Pre-operative adiogram 5-2-55. Post-operative audiogram 8-5-55.

Case 2. R. W., No. 736200, a white male, aged 14, was admitted to the University of Michigan Hospital on Sept. 20, 1952, with an acute head injury. Earlier the same day a jack had slipped, allowing the car on which he was working to fall on his head. Examination revealed a partial left peripheral type of VIIth nerve palsy, paralysis of the left Vth and VIth cranial nerves and of the right second and third cranial nerves. Radiographs of the skull demonstrated multiple fractures on the right side and a fracture on the left side extending from the left tempero-parietal area into the mastoid (see Fig. 7).

During the next four days the facial paralysis became complete. Otoscopic examination revealed a deformity of the left external auditory canal in its supra posterior aspect. The left tympanic membrane was dull but intact. Tuning fork and audiometric tests demonstrated a mixed type of deafness on the left side with an air threshold averaging 53 db loss for the speech frequencies.

On Oct. 30, 1952, the facial paralysis was still complete, and there was complete reaction of degeneration (see Fig. 8). Electromyography indicated complete denervation of the left facial muscles. Testing with Faradic current up to 40 volts failed to elicit any facial response. Decompression of the facial nerve was advised.

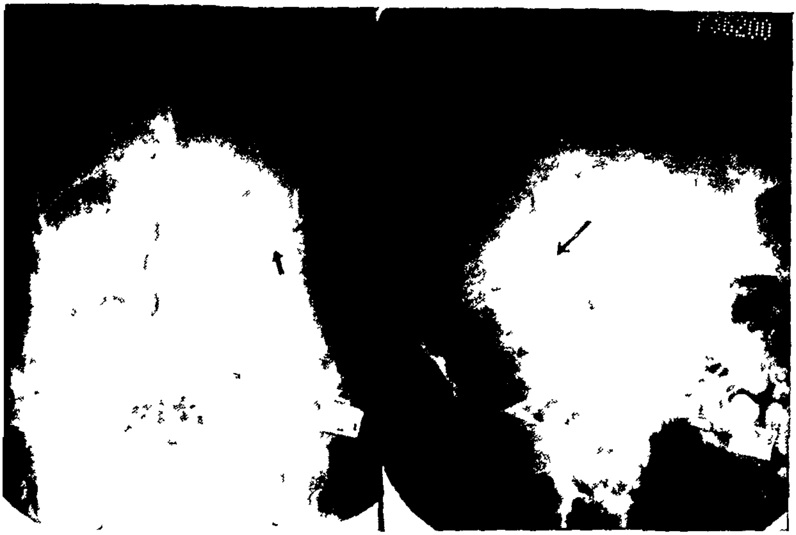


Fig 7 Case 2 Roentgenograms showing Fracture line extending from ft temporoparietal area into the mastoid Fracture involving right orbit

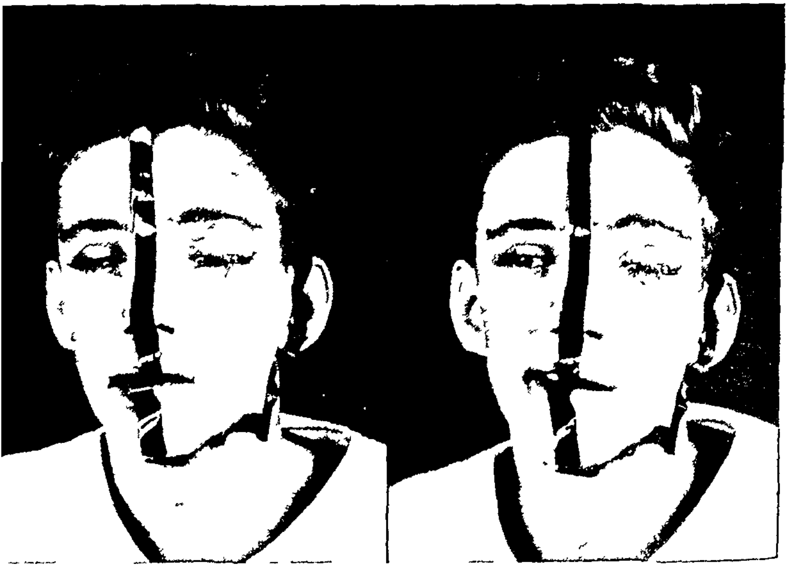


Fig 8 Case 2 Pre-operative photograph demonstrating left facial paralysis

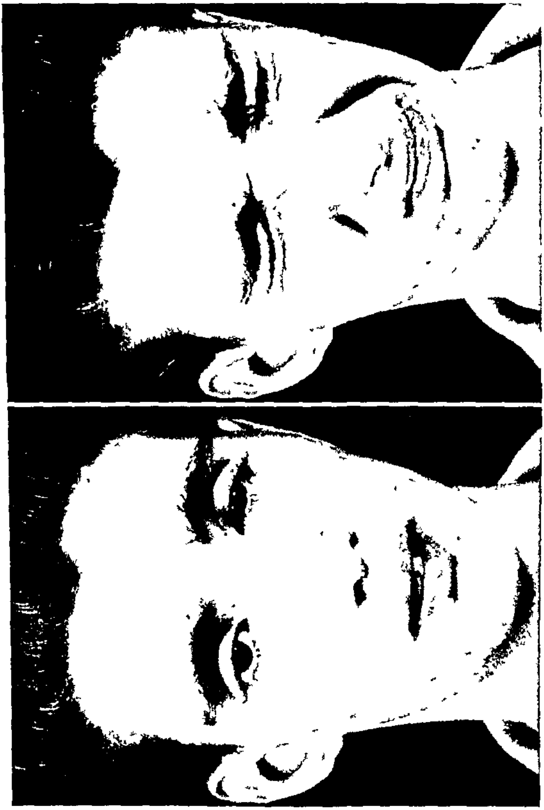


Fig 9 Case 2 Post-operative photograph, 9-2-55 Note *that tarsorraphy on left is still intact*

At operation, performed on Oct. 31, 1952, a fracture in the left temporal squama was found extending into the mastoid bone and through the posterior superior aspect of the external auditory canal. The fracture also extended through the pyramidal segment of the Fallopian canal, then through the tegmen of the middle ear lateral to the geniculate ganglion, and then forward toward the petrous apex. The incus, which had been dislocated anteriorly by the injury was removed. After the completion of a modified radical mastoidectomy, decompression of the facial nerve was accomplished by slitting its sheath. The nerve appeared to be normal from the stylo-mastoid foramen up to the pyramidal segment, but in the intratympanic portion it was grossly edematous and bulged through the opened sheath.

The patient had an uneventful convalescence and was discharged from the hospital on the eleventh postoperative day. Four weeks after operation facial movements were first noted and gradually improved to a maximum during the next four weeks. His most recent examination on Sept. 2, 1955, indicated good symmetry of the face in repose. He was able to close both eyes tightly and to show his teeth, purse his lips, and wrinkle the forehead on one side as well as on the other (see Fig. 9). There was minimal mass motion and a slight spasmodic tic of the upper and lower lip on the operated side. An audiogram on this date showed an air threshold of 31 db average loss for the speech frequencies in the left ear (see Fig. 10). The mastoid cavity was healed and dry.

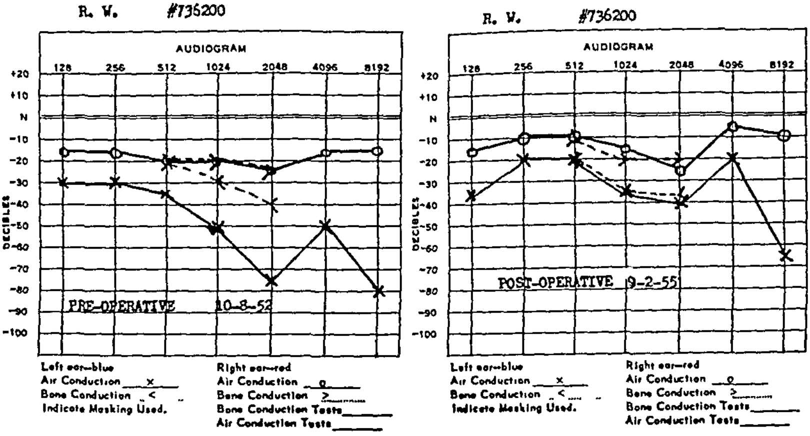


Fig. 10. Case 2. Pre-operative and post-operative audiograms.

Case 3: L. M., No. 811204, a white male, aged 34, entered the University of Michigan Hospital on May 30, 1955, because of a left facial paralysis. He had been well until Dec. 17, 1954, when he fell about 12 feet from the top of a boiler on which he was working. He struck a scaffold, fracturing his left shoulder and then struck the left side of his head on the floor, which caused a skull fracture. It was reported that he was unconscious for about five days and that he bled from the nose, the left eye, and the left ear. There was no history of cerebro-spinal otorrhea. The patient was told that the paralysis of the left side of the face was immediate Since the accident the patient had been deaf in the left ear but had not noted any dizziness

Routine examination of the pharynx nose and right ear revealed no significant pathological changes There was no paralysis of the tongue

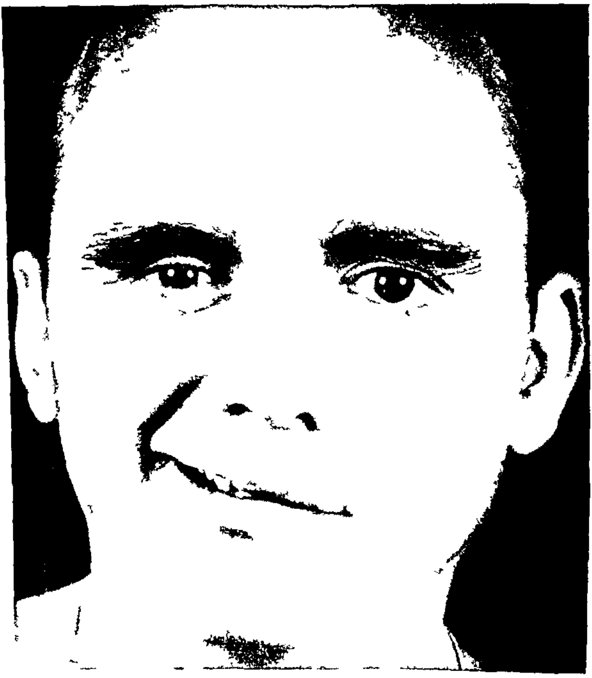


Fig 11 Case 3 Pre operative photograph showing left facial paralysis and obvious tearing of left eye

palate or larynx There was a deformity of the left external auditory canal appearing as an angular irregularity in its posterior aspect at the annulus where there was a little granulation tissue but no gross purulent exudate The patient had a complete peripheral left facial paraly and no response of the facial musculature could be elicited with 40 vo of Faradic current applied through the skin (see Fig. 11) The left eye

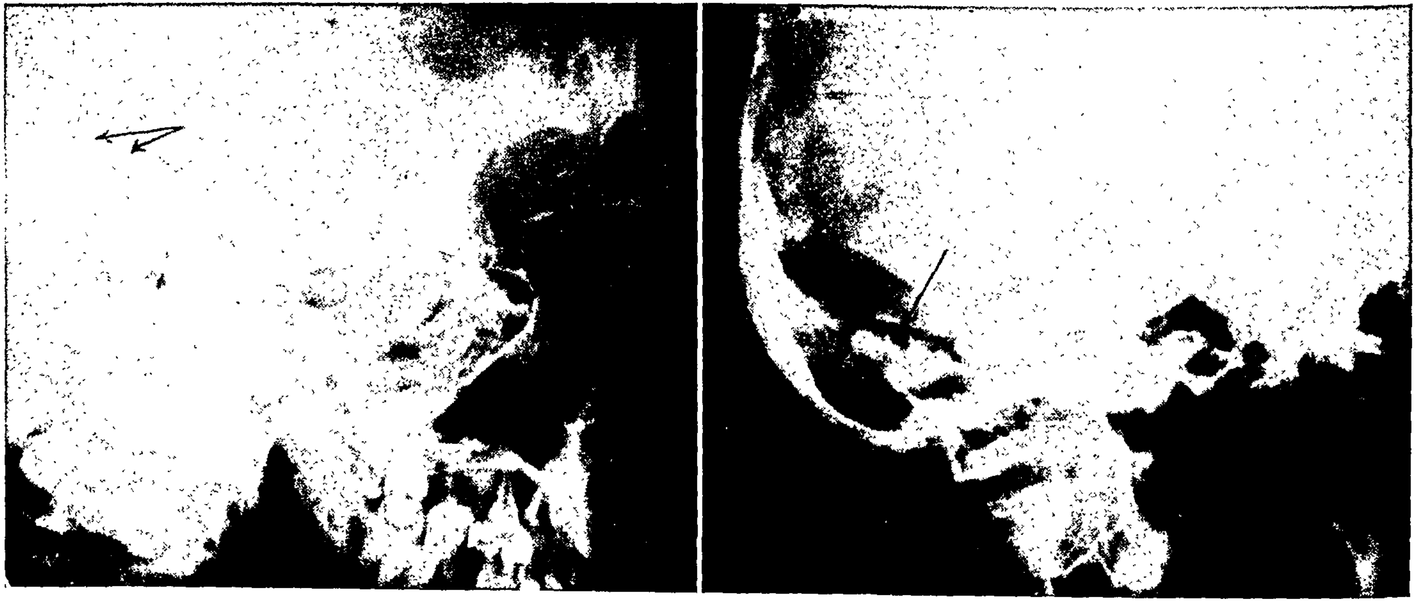


Fig. 12. Case 3. Roentgenograms demonstrating the fracture in the left parietal bone and the fracture line in the left petrous.

was moist, and the Schermer test showed lacrimation in the left eye that exceeded that in the right. There was also a loss of taste on the anterior two-thirds of the tongue on the left side. Functional tests demonstrated normal hearing on the right side, but total deafness on the left. No labyrinthine response was obtained after 40 cc. of ice water had been in-

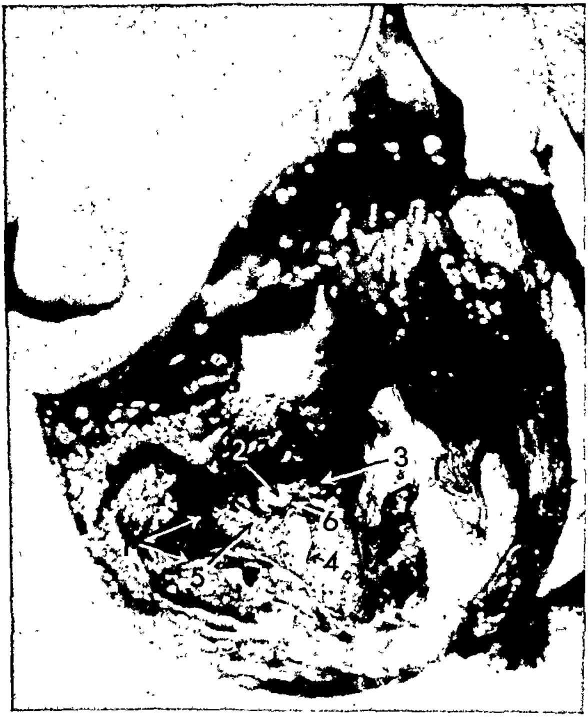


Fig. 13. Case 3. Photograph taken during operation at completion of radical mastoidectomy showing fracture through the horizontal semicircular canal.

Arrows indicate: 1. Mastoid tip; 2. Promontory; 3. Epitympanum; 4. Open fracture line across horizontal canal; 5. Exposed vertical course of facial nerve; 6. Mass of scar in region of intra tympanic portion of facial nerve.

jected into the left external canal during a period of 40 seconds. There was neither spontaneous nystagmus nor past pointing.

X-ray studies demonstrated a comminuted fracture of the left parietal bone without depression, and a fracture line which extended into the left petrous bone (see Fig. 12).

Because of the complete paralysis of the face of over five months' duration, which had been apparent immediately after the accident, and because of the marked tearing in the left eye and the deformity of the bony external auditory canal, it was felt that the facial nerve had been

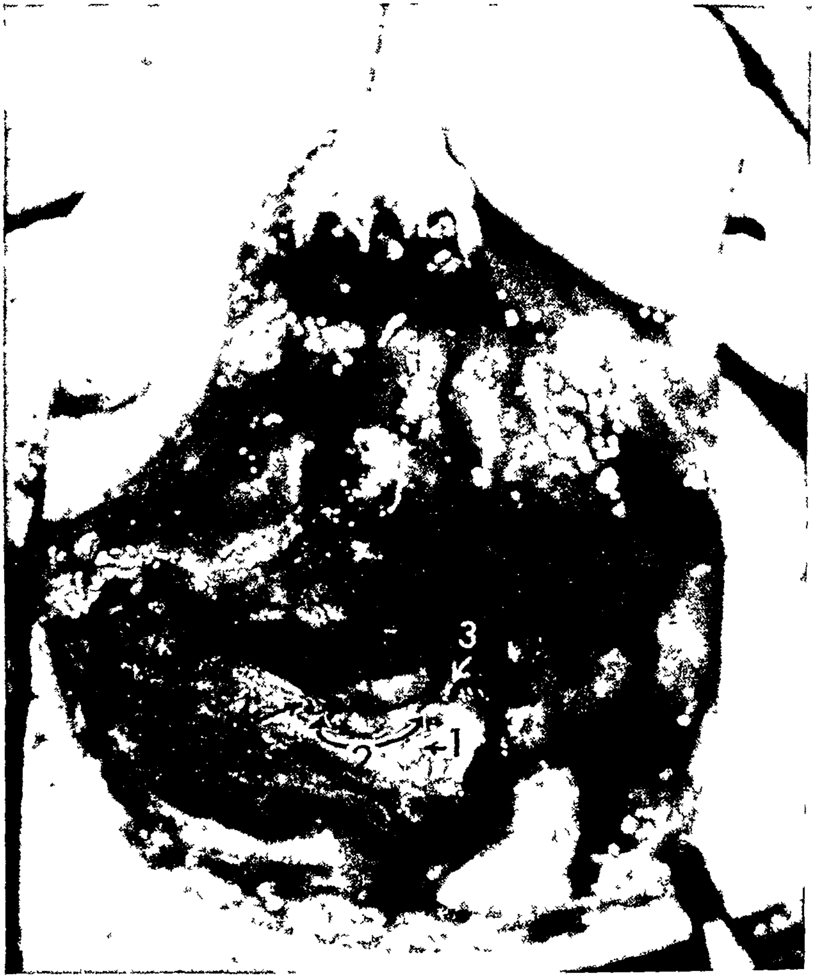
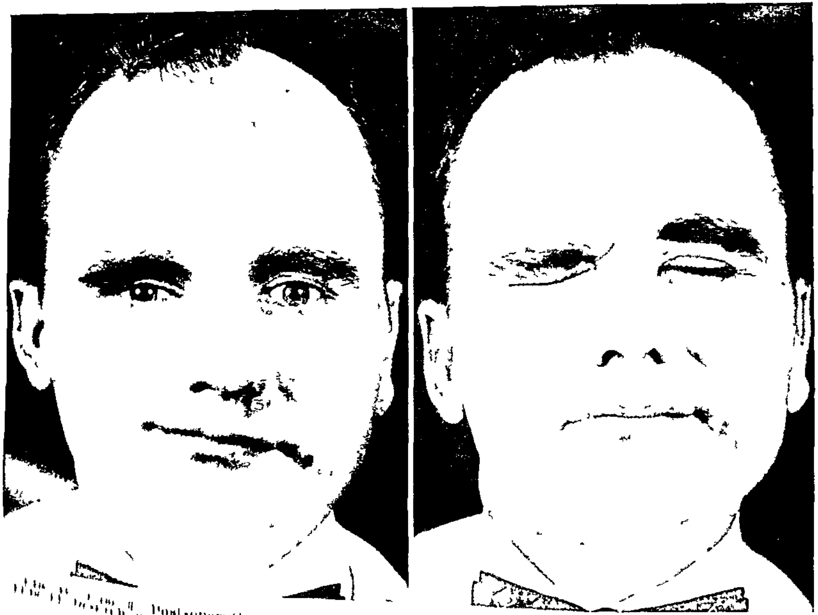


Fig 14 Case 3 Photograph at completion of operation showing nerve graft sutured in place

Arrows indicate 1 Fracture site across horizontal canal, 2 Nerve graft, 3 Proximal stump of facial nerve, 4 Distal stump of facial nerve



[illegible] Postoperative photograph taken on March 5 1956 (9 months after operation) Note [illegible] was added two months after presentation of this paper injured at a point distal to the geniculate ganglion in spite of the obvious fracture through the otic capsule, the total deafness and the loss of vestibular function.

On June 1, 1955, a left radical mastoidectomy was performed. It was found that the patient had a large pneumatic mastoid, which contained old blood but was free from any evidence of suppuration. Two widely separated fracture lines were noted in the temporal squama. The fracture had extended into the tegmen of the mastoid, where there were spicules of bone lying attached to discolored dura. As the antrum was excavated, a wide fracture line through the horizontal semi-circular canal and into the vestibule was visualized. The region of the intra-tympanic segment of the Fallopian canal was occupied by a mass of scar tissue. After completing the radical mastoidectomy, scar tissue in the region of the intra-tympanic portion of the facial nerve was removed, and it was found that the nerve had been severed just distal to the geniculate ganglion (see Fig. 13). A small stump of rather normal appearing nerve was preserved at this point, and the distal stump of the nerve was found just below its pyramidal segment. The ossicular chain had been disrupted, and all the ossicles including the displaced stapes were found in the middle ear. A 2 cm. free graft from the great auricular nerve was used to bridge the dehiscence in the facial nerve (see Fig. 14). Application of this graft was difficult because of the extremely short stump at the geniculate ganglion.

The patient's postoperative convalescence was uneventful. The wound remained clean and free from evidence of suppuration.

At the time of the patient's last examination, on Jan. 3, 1956, the ear was dry. The patient stated that his face had felt slightly different during the past month and that he was having less difficulty in talking. On attempted forceful closure of the eyes, the corner of the mouth would elevate. Apparently he was experiencing the first bit of functional return since operation, which was seven months prior to this examination.

SUMMARY.

Case reports of three patients suffering from complete peripheral facial paralysis due to middle fossa fractures have been presented.

One, Case 1, had nearly normal hearing pre-operatively. The operation was a simple mastoidectomy with removal of bone fragments at the pyramidal segment of the facial nerve, and decompression of the nerve from the geniculate ganglion to the stylo-mastoid foramen. Decompression of the intra-tympanic portion of the nerve was accomplished after further elevating the displaced short crus of the incus and deepening the fossa incudis to create space through which to operate. The postoperative hearing is nearly normal. Although complete return of function of the facial muscles did not take place, there was marked improvement, which began five days after operation and had reached a fairly satisfactory level two months later.

In Case 2, the patient had multiple cranial nerve palsies and a delayed facial paralysis. The longitudinal fracture apparently extended to the region of the Gasserian ganglion. Preoperatively, there was a mixed type of deafness with 53 db loss in the speech frequencies. The operation was a modified radical mastoidectomy, and decompression of the facial nerve. Return of function of the facial muscles began about four weeks postoperatively. The patient continued to have perceptive hearing loss of 31 db in the speech frequencies, but there was considerable improvement over the preoperative level.

Case 3 was that of a patient who had a fracture through the labyrinth and cochlea. The facial nerve was severed in its intratympanic course. Repair was accomplished after debridement of the nerve ends and the use of a 2 cm. free graft from the great auricular nerve. A radical mastoidectomy was required. At the time of this writing, January 4, 1956, just seven months postoperatively, the patient is showing his first signs of return of function by his ability to move the corner of his mouth slightly. There appears to be better tone in the facial tissue, and the patient has noted less difficulty with speech during the past month. If an excellent result were to be obtained, I believe that the first signs of returning function would have appeared earlier.

It is generally recognized that the prognosis for complete, or at least functionally adequate recovery from facial paralysis due to head injury, is good in the vast majority of cases. In all probability, surgical intervention need be considered in less than 10 per cent of such patients. Such consideration must include careful and complete study to determine, insofar as possible, the severity of the nerve lesion and its surgical accessibility. In properly selected cases, the results of operation to repair damage to the facial nerve will be beneficial and gratifying. If the preoperative hearing is serviceable, every effort should be made to preserve it. A radical mastoidectomy is not necessary in every case in which operation is required.

ADDENDA.

L. M., No. 811204, the patient described in Case 3 was examined on March 5, 1956, at which time returning function was noted (see Fig. 15) and further improvement is likely.

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AN OTOLARYNGOLOGIC APPROACH TO MALIGNANT EXOPHTHALMOS.*

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It is not the purpose of this paper to deal with the etiology or pathology of progressive or malignant exophthalmos, but rather to review the historical background of the surgical relief of the condition and to bring again to the profession a rhinological approach to the problem. Progressive, or malignant, exophthalmos is a serious disorder unless controlled by medical or surgical therapy. Not only will it cause blindness, if progression goes on, but also corneal ulcer, ophthalmitis, and even meningitis and death.

The first operative procedure which I have been able to find in literature was reported by R. Foster Moore[1] in 1920. He made an incision through the whole length of the inferior conjunctival fornix, exposed the contents of the orbit, and with forceps picked away piecemeal as much of the orbital fat as possible. He estimated that a heaped-up teaspoonful was thus removed. Afterwards, the lids were easily approximated and were in part sewn together over the cornea, as also were the left lids, and by this means both eyes were preserved.

In 1930, O. Hirsch[2] reported a decompression of the orbit for progressive exophthalmos by removing the floor of the orbit, using the canine fossa approach and entering the antrum of Highmore; then removing the floor of the orbit, leaving only a narrow strip of bone containing the infraorbital nerve, thus protecting the nerve. He also states that to give more room, this bone and nerve may be removed, but loss of sensation or hypesthesia of cheek will ensue. We have never done this type of operation, but in talking to Theodore Walsh of Washington University School of Medicine, he stated that he

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did one through the canine fossa, but that it did not work, and he had to do a Sewall type decompression.

It was not until 1932 that Naffziger and Jones[3,4] reported their surgical treatment of progressive exophthalmos following thyroidectomy. At that time, they reported six persons who had been operated upon by their method. In these articles they established the principal etiological factor in this condition. The Naffziger operation is an operation performed by neurological surgeons and is described thoroughly in these articles. They were the first to explain the pathology of the condition, and their histologic sections confirm their opinion. The method they employ consists of entrance to the skull through the frontal region by means of a transverse incision from the temporal fossa of one side to the temporal fossa of *the other side, going across the fronto-parietal region immediately* behind the hairline. The scalp is dissected from the pericranium, and is reflected down, exposing the entire frontal bone on both sides. down to the frontal sinus. Bilateral frontal flaps are then fashioned, the hinge of each being the temporal muscle. A strip of bone is left in the center to stabilize the bone flaps when replaced. The dura is now separated, with a broad spatula, from the floor of the anterior fossa and down the sphenoidal ridge and the base of the anterior clinoid. Then the roof of the orbit is removed and the optic nerve is decompressed by removing the roof of the optic foramen. When this is finished, the orbital fascia is opened, being careful not to cut the frontal branches of the Vth nerve. The entire bony roof of the orbit is then removed. (See Fig. 1.)

In 1935, Swift[5] of Seattle, Wash., reported on Malignant Exophthalmos and Operative Approach. He reported one case on which he did a modified Kroenlein operation with good result.

In 1936, Sewall[6] read a paper before the Western Section of the *American Laryngological, Rhinological and Otological Society,* on Operative Control of Progressive Exophthalmos, and this was published in the *Archives of Otolaryngology.*

In 1939, Kistner[7] reported two cases operated upon by this method and one case by the Naffziger method.

In 1945, Schall, L. A., and Reagan, D. J.,[8] reported two cases operated upon by the Sewall method. In their conclusions they state, "Two cases of malignant exophthalmos are presented that were treated by decompression into the sinuses following the method proposed by Sewall and first used by Kistner. We agree with Kistner's conclusions that this operation is:

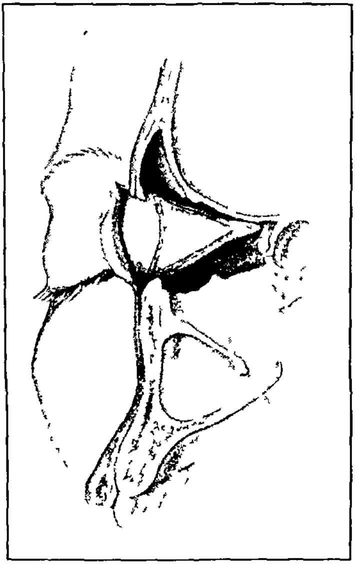

Fig 1

1. Less disfiguring and formidable than the Naffziger operation.

2. An actual rather than a potential space is created.

3. Sometimes the roof of the orbit is occupied by extension of air cells which the Naffziger technique avoids entering.

4. There is no subjective or objective evidence of pulsation in the orbital contents which may follow the intracranial operation."

In the Sewall[9] type of operation, we use the fronto-ethmoid technique which was described by him in November, 1926, in the *Archives of Otolaryngology.* Anyone who has done this type of operation and removed the lamina papyracea has seen the orbital contents bulge into the nasal cavity, and this is especially true if during the operation a tear has been made into the capsule and fat of the orbit protrudes. It is this fact, I am sure, that led Sewall to advocate this type of operation for the relief of progressive exophthalmos. It is possible in this type of operation to remove the three walls of the orbit; also, if need be, one may decompress the optic nerve by removing the lateral margin of the optic foramen. In this type of procedure, one can remove supraorbital ethmoid cells if they are present. After the lamina papyracea has been thoroughly removed, at least as far back as the sphenoid sinus, and the floor of the frontal sinus is entirely removed, the ethmoid labyrinth exenterated and the lining of the frontal removed, incisions are made by opening into the orbital fascia. Kistner[7] described three incisions: two anteroposterior incisions, and one transverse incision. The anterior posterior incisions are placed, one at the lower border of the resected lamina papyracea, the other at the upper border. The transverse cut is started at the external limit of the frontal sinus and carried internally across the anterior ends of the two previous incisions. Before closing, the mucoperiosteal flap should be placed to protect the nasofrontal opening as described by Sewall.[9]

We report two cases which are interesting and instructive.

Case 1: W. H. D., male, age 31, truck driver: First seen June 24, 1947, and stated the proptosis was first noticed two months previously. Weight loss eight pounds past six months; appetite very good. No tremor noted except when hungry. Examination showed marked exophthalmos of both eyes, but right eye worse, inability to close lids, showed the stare peculiar to exophthalmos. Proptosis 24 mm. right eye, 21 mm. left eye, October 10, 1947. There never was any enlargement of right or left lobes of thyroid, or of the isthmus. No substernal thyroid. Laboratory findings all negative except basal metabolic rate which went to +44. Diagnosis: Pituitary-thyroid progressive exophthalmos. Large corneal ulcer on left. Left side decompression done November 6, 1947; right side decompression done November 20, 1947. Uneventful recovery; well to present time.

Case 2: Mrs. E. W. Mc., age 60. This white female entered Emanuel Hospital August 25, 1953, after having been released from the hospital one month previously, at which time a thyroidectomy was done. Since discharge, she had had a persistent pain in both eyes, headaches over eyes, and especially the left. Exophthalmos progressively getting worse. Physical examination showed blood pressure 146/96; eyelids were sutured together and bandaged. Evidence of thyroidectomy scar. Eyes checked by Dr. U'Ren, an ophthalmologist. The diagnosis of progressive malignant exophthalmos was made, with intense retro-orbital and periorbital edema. Both corneas were ulcerated, particularly the left. On August 27 a diagnosis was made of left endophthalmitis with eyeball full of pus. On August 29 an enucleation of left eye was done. The patient was referred to us on September 29, 1953, with a recommendation for a decompression operation of right eye. On examination, we found a deep ulceration of about half of the cornea. On October 3, 1953, a decompression operation was done on the right eye. When through with the operation, she could close her lids. On the sixth day the ulcer was healed. No doubt the left eye could have been saved if seen sooner. Her vision is poor, due to the corneal scarring.

In conclusion, we advise an early decompression for progressive exophthalmos in order to save eyesight and possible death from meningitis.

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ANNUAL ASSEMBLY IN OTOLARYNGOLOGY.

The Department of Otolaryngology, University of Illinois College of Medicine, announces its Annual Assembly in Otolaryngology from October 1 through 7, 1956. The Assembly will consist of an intensive series of lectures and panels concerning advancements in otolaryngology, and evening sessions devoted to surgical anatomy of the head and neck, and histopathology of the ear, nose and throat.

Interested physicians should write direct to the Department of Otolaryngology, 1853 West Polk Street, Chicago 12, Ill.

APC VIRUSES IN RESPIRATORY DISEASES—CLINICAL ASPECTS.*

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Respiratory diseases of apparently non-bacterial origin have been a perennial problem to those dealing with the illnesses of children, of military recruits and of industrial workers.

May I review with you, certain recent advances in our knowledge of these diseases, mainly the work and experience of Drs. Rowe, Huebner and Bell, at the National Institute of Health, and Dr. Ward, at Johns Hopkins.

The story as I will review it,[§] in a sense, had its beginning with physicians in your field. In 1952 and 1953, tonsillar and adenoid tissues were collected from children undergoing surgery at the Children's Hospital in Washington, and at several other hospitals. These tissues were minced up and placed in test tubes for tissue culture in the hope that they would grow *in vitro* and provide a host for the culture of common respiratory viral agents. Instead, over 50 per cent of the tissues themselves were found to contain viral agents which could then be transmitted serially through other tissue cultures.[1] These viruses and a group of closely related agents are now named the adenoidal-pharyngeal-conjunctival viruses for which the abbreviation APC is used. At about the same time Hilleman isolated, from cases of acute respiratory disease in military personnel, a virus called RI-67, which shares the characteristics of the APC group.[2] This group of APC viruses includes the cytopathogenic agents which were originally isolated from tonsils and adenoids and have subsequently been

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* Revised transcription of a paper read before meeting of the Eastern Section, American Laryngological, Rhinological and Otological Society, Inc., Boston, Mass., January 13, 1956.

† From the U. S. Department of Health, Education and Welfare, Public Health Service, National Institutes of Health, National Institute of Allergy and Infectious Diseases, Bethesda, Md.

§ Parts of this talk have been published previously in Clinical Proceedings of Children's Hospital, Washington, D. C., 11:152-158, 1955.

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found in conjunctival, pharyngeal and fecal material.[1] The human sources of these viruses indicate why the name was proposed: they were first isolated from adenoid tissue; human conjunctival and pharyngeal material have been the major sources of these viruses subsequently. These agents are not pathogenic for laboratory animals. They do show a unique destructive effect in tissue culture of epithelial tissues, particularly of the serially propagated HeLa cell strain of human cervical carcinoma. At least some of the strains cause similar effects in monkey kidney and in human embryo fibroblast in tissue culture. The viruses are ether resistant, heat labile and filterable. They resist antibiotics. Members of the group share a group specific soluble antigen which gives a group reaction in a complement fixation test. This means that by a complement-fixation test these types cannot be differentiated. The agents do produce a type specific neutralizing antibody and on the basis of neutralization tests it can be said that there are at least 10 or 11 immunologically distinct types of APC viruses.[4]

Serological studies, mainly neutralization tests, indicate that over 50 per cent of infants in the 6- to 11-month age group show evidence of having been infected with at least one type of APC virus.[5] There is a sharp rise in evidence of infection from ages one to two. There is a less prominent increase in the number of individuals with neutralizing antibodies from ages three to five. Gradually thereafter, there is increasing evidence of individuals having been infected with at least one of the virus strains.

What do these viruses do clinicall
available yet concerning the clinic
APC virus types. Two well studie
for which the name pharyngo-conju
have provided reasonably adequate
the etiological associations and the
APC infection.[5,6] Evidence that phar
is etiologically related to Type 3 AP
facts derived from an epidemiological
agents invaded the tissues of persons w
in other words, the individuals showed
response; 2. they were found in the site

sion in the clinical entity, *i.e.*, found in the throat, if the throat was infected; in the eye, if the eye was infected; *3.* they were not present in well persons who were in intimate contact with the individuals who had the illness; *4.* the viruses were present in the acute stage of illness, not before and not after; *5.* they were associated consistently with a particular clinical entity; and *6.* they were not found in other illnesses occurring at the same time as the outbreak studied or in sporadic studies of many cases of respiratory and other kinds of illnesses.[6]

In addition, recent inoculation of these agents in human volunteers has reproduced essentially the clinical entity which I will describe.[7] It is interesting to observe that originally, the inoculation of these agents into the nasopharynx did not produce clinical illness, but when the material was inoculated into the conjunctiva, the characteristic illness was reproduced.

The first outbreak of Type 3 APC virus infection associated with a clinical illness (pharyno-conjunctival fever) occurred in a hospital ward and included eight patients. In these patients, pharyngitis was a common complaint and finding. Two patients had exudative pharyngitis. Rhinitis was a very frequent symptom and sign. High fever lasted for five to six days. In six of the patients, cervical lymphadenopathy and conjunctival erythema were noted.

One youngster in this outbreak may be interesting to your group, particularly as she had a complicating sinusitis and otitis. Most of the other individuals with this infection whom we have seen during the summer months do not have these complicating features. The patients whom we have seen during the winter months seem more likely to have paranasal obstructive disease.

There was lack of severe leukocytosis and toward the end of the illness, there was relative leukopenia, with a slight lymphocytic predominance.

Table I summarizes the symptoms and signs that occurred in a much larger group of individuals in a summer day camp outbreak of this disease.[6,9] The symptoms included fever, complaint of sore throat, red eyes, headache, malaise, and occasionally aching and a general feeling of malaise which was

TABLE 1

MAJOR FEATURES OF PHARYNGO CONJUNCTIVAL FEVER
IN A CAMP OUTBREAK

SYMPTOMS	Frequency
Fever	90%
Sore Throat	70%
Red Eyes	66%
Headache	68%
Malaise Weakness	72%
SIGNS	**Frequency**
Fever 102° 104° for 4 5 Days	90%
Pharyngitis	75%
Cervical Adenopathy	75%
Posterior	66%
Rhinitis	50%
Conjunctivitis	75%
Unilateral	25%
Bilateral	50%

more common in older people, such as the parents of the children involved Cough was not really a prominent symptom Vomiting, diarrhea and epistaxis occurred in a few of the patients High fever, lasting four or five days, occurred in almost all the patients Pharyngitis consisted predominantly of hyperplasia and erythema of lymphoid tissue on the posterior pharynx and on the anterior pillars of the tonsillar fauces Lymphadenopathy was distributed most strikingly in the posterior cervical areas Rhinitis was manifest by rhinorrhea and by mild inflammation Conjunctivitis affected one or both eyes, in some instances the conjunctivitis was unilateral initially, and became bilateral Both the bulbar and palpebral conjunctiva were affected with superficial inflammation that was manifest mainly by injection of vessels There was no pruritis, but there was a great deal of watery exudation A few patients showed signs of meningismus A few had cough, and ronchi could be detected

The four major clinical features, then, of pharyngo-conjunctival fever are fever, conjunctivitis, pharyngitis, and posterior cervical lymphadenopathy There may be several combinations of these findings in different patients

Pharyngo-conjunctival fever occurs epidemically and sporadically[6] Epidemicity is probably defined by the degree of immunity existing in the community or group attacked. It affects all ages, all sexes, although mostly children again, because of their relative susceptibility. Among the adult contacts the mothers were more frequently infected than the fathers. This probably also represents a matter of opportunity to have been infected previously. Virus is spread by direct contact almost surely, and probably by respiratory secretions.

The usual portal of entry is somewhat in question because of the points brought up by the human volunteer experiments. It is hard to know what is happening in natural infection if in an experimental infection one cannot induce the disease by nasopharyngeal inoculation. There is a very high attack rate among susceptible individuals; in children four to seven years of age, for example, the attack rate was 72 per cent in the summer day camp. There is a low or absent carrier rate; no individual who did not have the clinical syndrome showed evidence of infection with the virus. There is evidence of advancing immunity with age. The incubation period is five to six days. The period of communicability is up to nine days. That swimming pools play a role in the spread of this disease is suggested by the several instances in which outbreaks occurred among persons having contact with camp or neighborhood pools.[6,10,11] Demonstration of virus in the water has not been possible. Two possible explanations for the apparent association of pharyngo-conjunctival fever with pools come to mind: *1.* the pools are a congregating place during the summer and congregation facilitates direct spread; *2.* swimming in pool water causes conjunctival irritation and allows ready ingress for virus spread by contact with other persons.

Table II summarizes what we presently know about the sources of the other APC virus types. It may be of particular interest to your group to know that all of the Types 1 through 6 have been isolated at one time or another from tonsillar and adenoid tissue. Types 1, 2, and 5, have been found frequently, not only by our group but also by other laboratories, in pharyngeal lymphoid tissue removed at surgery.[12] The question of what the viruses may be doing in those tissues is one for which I think we have no real answer. One hypothesis is that persons undergoing acute infection with these agents

TABLE II.

RESUME OF HUMAN SOURCES OF APC VIRUSES.

APC TYPE	1, 2, 5	3	4	6	7	8	9	10
Tonsils, Adenoids	+	+	+	+				
Febrile Pharyngitis	+	+						
Pharyngo-Conjunctival Fever		+						
Acute Respiratory Disease			+		+			
Kerato Conjunctivitis						+		
Catarrhal Conjunctivitis				+				+
Poliomyelitis Survey							+	

carry the viruses at least for periods of time, in the pharyngeal lymphoid tissue. In partial support of this hypothesis is the fact that in the Washington, D. C., area, for example, Type 3 virus had never been isolated from a tonsil or adenoid until the year when there was a widespread outbreak and widespread occurrence of Type 3 in acute disease. The next year a few tonsils and adenoids had Type 3 in them.

Types 1, 2 and 5 have also been found in less specific febrile pharyngitis in children in studies which were not so well controlled clinically as those I have mentioned.[3,13] Type 3 has also occurred in a few cases of febrile pharyngitis,[3,14] and in a disease resembling roseola infantum.[15] Type 4 virus, known also as Respiratory Illness 67, or RI-67 virus by Hilleman, has been found as "recruit disease," what the Commission on Acute Respiratory Disease has called ARD, and in primary atypical pneumonia-like illness almost exclusively in military recruits, Army and Navy.[2,16,17,18,19] Type 6 has been found in two patients with catarrhal-conjunctival infection and mild respiratory symptoms.[10] Type 7 has been found in distribution similar to Type 4.[14] Type 8 has been found in cases of epidemic keratoconjunctivitis in industrial workers.[21] Type 10 has been found in a case of catarrhal conjunctivitis,[4] Types 9 and 11 were isolated from feces in a survey of patients with poliomyelitis-like disease.[4,22]

There remain many problems in this field. First of all, are similar groups of viral agents responsible for many other respiratory diseases so often seen in practice? Second, concerning this very prevalent group of APC viruses, there are many questions which one might ask, for example, what are they doing in the tonsils and adenoids? Could they be associated with the process of adenoid and tonsillar enlargement? What other clinical entities might be associated with APC virus infection? What therapy would be indicated for infection with these agents? In our experience no antibiotic or chemotherapeutic agent seemed to have a therapeutic effect in the patients studied for the acute uncomplicated illness. Those few children who had complicating otitis or sinusitis did seem to respond to antibiotic therapy; it was presumed in these cases that obstruction of the paranasal orifices allowed bacterial infection to occur.

Another most provocative question is: What are the possibilities of immunizing human beings against these agents? The viruses are very prevalent. There is evidence that they are responsible for a large amount of respiratory disease. In volunteers, some vaccinated individuals resist challenge with the type virus in the vaccine.[23] The agents can be grown readily in tissue culture, and a vaccine has been made and could be made on a larger scale. It is not too unlikely that an effective vaccine might be developed which would eliminate a significant amount of respiratory disease.

In summary the APC, or adenoidal-pharyngeal-conjunctival viruses, are newly isolated epitheliotropic tissue culture agents. They have been associated predominantly with an illness named pharyngo-conjunctival fever and with respiratory disease in military recruits. If the major features of pharyngo-conjunctival fever, conjunctivitis, pharyngitis, and cervical lymphadenopathy are present, or a combination of them is present in the face of an epidemic, I think one can be fairly definite about the clinical diagnosis of Type 3 APC infection without having a virological diagnosis made.

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SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Arrangements have been completed for the joint meeting of the North Carolina Society of Eye, Ear, Nose, and Throat, and the South Carolina Society of Ophthalmology and Otolaryngology September 17, 18, 19, 1956. Headquarters will be the George Vanderbilt Hotel, Asheville, North Carolina.

An unusually attractive program has been arranged, and a large attendance is anticipated.

Asheville, North Carolina, is in the mountains of Western North Carolina, and is a particularly beautiful spot in this season of the year.

For further information write Roderick Macdonald, M.D., Sec. and Treas., 330 East Main Street, Rock Hill, S. C.

A NEW METHOD OF BRONCHOSCOPY WITH CINEMATOGRAPHY AND PHOTOGRAPHY.*

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ROGER J. EDWARDS, M.D.,

and

ANDRE-J. GLADU, M.D.,

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A new French apparatus now permits an endobronchoscopic film to be produced in the course of each bronchoscopy. An objective record of the specialist's examination may thus be kept, a document which may be studied or discussed in the interest of the patient.

It would not occur to a radiologist to be content with a fluoroscopic examination when he detects a pathological condition of the lungs. The endoscopist, likewise, has felt for many years that his reported observations, often recorded in considerable haste and illustrated with a diagram (even when such a diagram is drawn with the greatest skill and accuracy), represents records which are still very incomplete.

Dr. Paul Holinger, of Chicago,[1,8,9] should be credited with having produced the first endobronchoscopic films. His films are known not only in America but throughout the world, as well. With his photographic equipment, endobronchoscopic 16 mm. color films of rare quality may be made at 16 or 24 frames per second. In France, Dr. André Soulas has used similar equipment, obtained in Chicago nearly 10 years ago, to produce the first European endobronchoscopic films.[10,11]

As a result of improvements recently achieved with a new bronchoscope developed by Fourestier, Gladu and Vulmière, accurate records may now be obtained through post-telescopic cinematography, at 16 frames per second, utilizing 8 mm.

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*Travail du Centre Régional de Lutte contre le Cancer (Directeur· Professeur Raymond Lefèvre), et du Service de Pneumo-Phtisiologie de l'Hôpital de Reims (Chef Pierre-M Dubois de Montreynaud, M D)

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films, which are not expensive, and recently 16 mm. films have become possible as well.[2,3]

The apparatus, of the usual diameter, is easily handled. For this reason, we have been able to do systematic cinematography in our service since June, 1954. Every bronchoscopic examination thus becomes the object for a film, yielding valuable objective data, which may later be studied at leisure and compared with the rest of the patient's record. This method gives a faithful reproduction of the bronchi, whether normal or pathological, and is applied in several Paris hospitals, especially in the Laënnec Hospital (Pr. Et. Bernard), in several province hospitals and particularly in the Reims hospital.

The bronchologist very quickly discovers the secrets which allow him to obtain excellent films. When the operator and personnel are accustomed to the technique, filming the examination requires no more time than a normal bronchoscopy; it permits excellent illumination during the bronchoscopy, and there is no delay in seeing the photographs, which can be inspected as often as desired once the film is developed.

The life-like character of the endobronchoscopic pictures is particularly interesting. The normal movements of the bronchi are: inspiratory enlargement of the bronchial caliber, ascending expiratory movements of the whole bronchial tree, transmission of heart beats, suppleness of the mucous membrane; also important changes observed in asthma, cancer, etc., or in experimental studies in animals, can be easily followed.

By this method the head of a large hospital service may note the state of patients' bronchi. Formerly this was possible only for the bronchoscopist. The same is true for assistants and students; the educational nature of these films cannot be too strongly stressed.

At the time of the medical-surgical conference, in an era when pulmonary or bronchial resection is essentially conservative, it is particularly important for the bronchoscopist or surgeon to have information which will be useful at the time of intervention.

Systematic endobronchoscopic cinematography will henceforth permit each patient to have documented observations which are actually complete. It is easy for the bronchologist to have a small projection room near his office where a projector is permanently installed. It takes no longer to run a film than to view a series of tomograms. Such endobronchoscopic documents have considerable interest, not only for the bronchologists, but also when presenting their work before professional societies, bronchial lesions can be shown as well as discussed.

DESCRIPTION OF THE APPARATUS.

It was necessary to find a simple means, available to every practitioner to combine classical bronchoscopy and photography in a simple operation, just as it is possible to pass from radioscopy to radiography within a few seconds, through the simple adjustment of a cassette.

Today the problem is solved, and we have an apparatus utilizing bronchoscopic tubes of the usual diameter, and the usual telescopes through which the pictures are taken, with illumination 200 times more powerful. Photography or cinematographic recording in every interesting detail may be made instantaneously with the simple aid of a photographic apparatus, or with any commercial camera desired, placed against the eye piece of the telescope.[6,7]

This apparatus is composed of several parts (see Figs. 1 and 2):

The bronchoscopic tube; a particular lighting indispensable for photographs or cinema, including a powerful light source and a conductor for that light, with an increased lighting coefficient.

Direct and lateral vision telescopes adapted to the Wolde bronchoscope and provided with an anti-vapor system, the importance of which is paramount in the success of photographs or films.

A number of parts, including an adjustable electric supply, an aspirator for cooling the lamp and a compressor for the anti-fogging system, all of which are grouped according to the models.

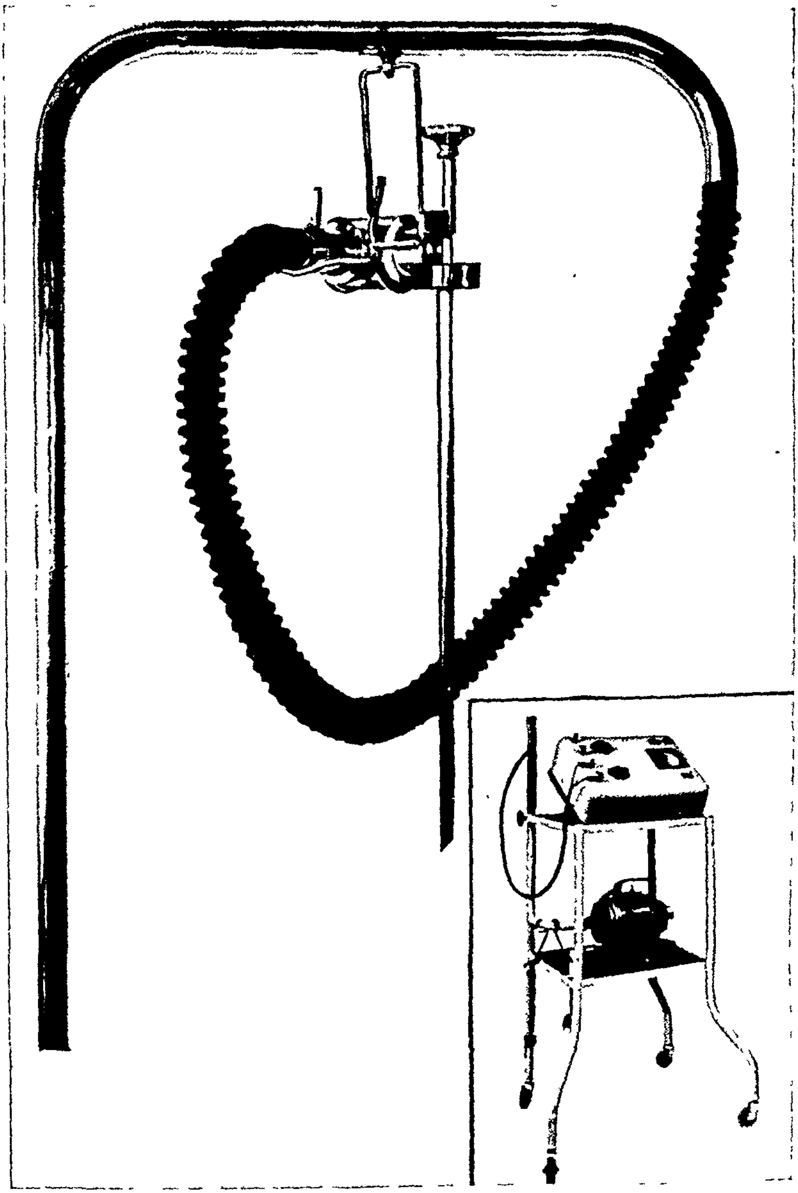


Fig. 1. Photography of the endoscope from Fourestier, Gladu and Vulmiere.

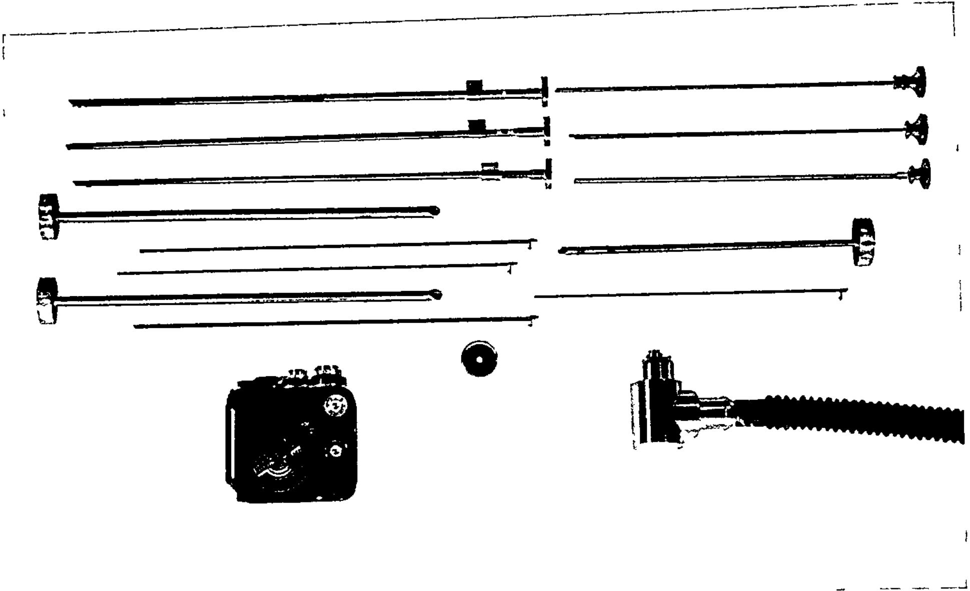


Fig 2 Details of the equipment A on the left for adults two tubes one direct telescope two right-angle telescopes two direct quartz-rods, one lateral quartz-rod B on the right for children one tube one quartz-rod and three telescopes, C below, the motion-picture camera, the adapter and the housing which contains the bulb

THE BRONCHOSCOPIC TUBE.

The bronchoscopic tubes present few modifications in relation to those of classical instrumentation.

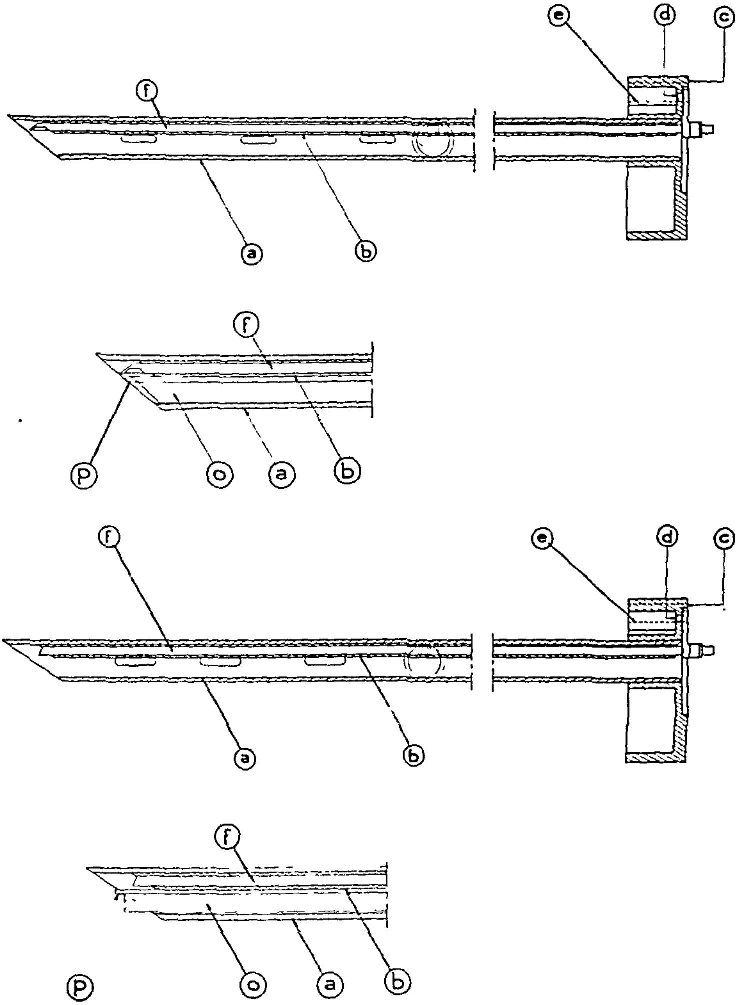


Fig. 3. A. the bronchoscopic tube; B. the light carrier-canal for the quartz-rod; F. the quartz rod; O. the telescope (direct or right angle).

The diameter and length of the tube for an adult are exactly the same as those currently in use, *i.e.,* 8 mm. diameter and 40 cm. in length; the thickness of its wall is 0.5 mm.[4]

The tubes must be made of an alloy with a nickel base instead of brass, so as to be perfectly rigid to avoid breaking the quartz rod, in the course of bronchoscopic maneuvers. This provides a much greater resistance to bending, which is very important if the larynx is anterior or the neck short (see Fig. 3).

The classical light carrier canal into which the quartz rod is introduced is soldered to the interior of the principal tube, so that the bronchoscope may be rotated in the bronchus without trauma. The light carrier canal has a diameter of 3 mm. and the wall is 0.1 mm. thick. The interior of the tube is covered with an electrolyte deposit of black, flat, non-reflecting nickel.

The distal extremity of the tube has a classical beveled aspect, but at its proximal extremity, a plate 50 mm. in diameter, permits the illuminator, Galileo magnifier or proximal telescope, to be adjusted or removed rapidly by means of pegs. We now have tubes for children, based on analogous principles.

LIGHTING.

The lighting constitutes the true originality of the system. Until recently in France a distal light source has been used. The illumination given by this small bulb, a masterpiece of manufacturing, nevertheless, is not comparable to that obtained with the new apparatus. A proximal light source is now used, the size and power of which is limited only by optical considerations. The image of the filament is transmitted to the distal end of the scope by a transparent rod of fused silicon (quartz-rod).

a. The light source is composed of a standard microscope-type lamp of 6 or 8 volts, 5 amperes, with a 2 mm. filament. This is placed in a metal case of a size which permits ease in manipulation. The lamp housing is a cylinder 90 mm. long and 50 mm. in diameter. Its weight is about 650 gr. It is cooled by air drawn through the lamp housing (see Figs. 4, 5 and 6).

The filament is centered in the housing in such a that it is at the optical center of a concave mirror. T and reflected light is then picked up by a condenser composed of two convex plane lenses with aspheric surfaces. The beams are then directed at a 90° angle by a prism to the exit of the housing, which consists of a small orifice 3 mm. in diameter.

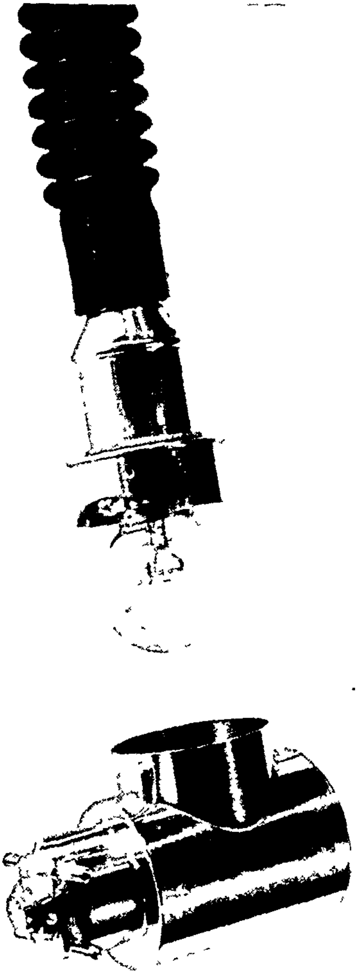
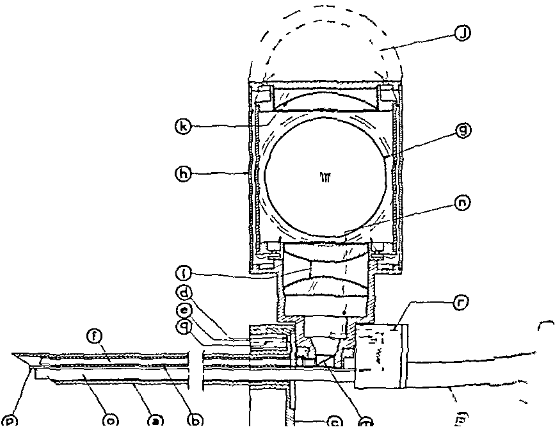
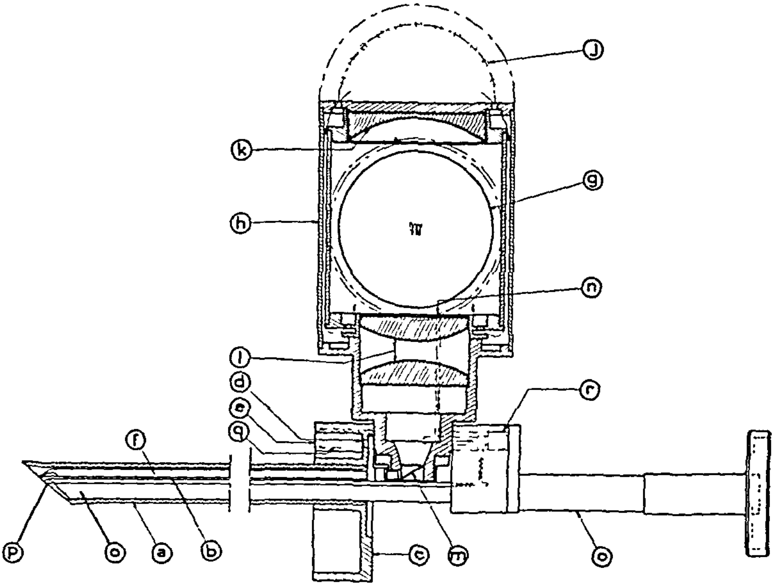


Fig. 4. The housing is open.

This orifice fits exactly against the proximal end of the quartz rod. When the lamp is illuminated, a special viewer permits an exact centering of the filament. The centering can be modified with the aid of three special screws which control the support of the bulb.

The lamp housing is cooled by air drawn through it. The air penetrates through 24 small holes in the bottom of the housing and escapes through a large tube on the side of the





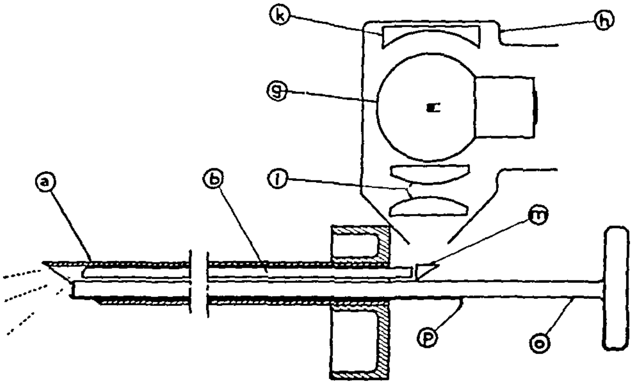


Fig. 6. The same diagram with right angle quartz-rod: A. the bronchoscopic tube; B. the light carrier-canal for the quartz-rod; G. the bulb; H. the housing; K. the concave mirror; L. the two convex plane lenses; M. the prism; O. the telescope; P. the small canal for air pressure for anti-fogging.

Simplified diagram: A. the bronchoscopic tube; B. the quartz-rod; G. the bulb; H. the housing; K. the concave mirror; L. the lenses; M. the prism; O. the telescope.

b. The quartz rod has a diameter of 2.5 mm. and is 410 mm. long. It is especially made, partially encased in silver and copper and surrounded by a protecting envelope of nickel. Its two extremities are smooth; the proximal end is cut at right angles, the distal end has a form which varies with the direction of the light tract. The rod is either beveled, to send the ray solely into the field of the direct telescope, or it terminates in a mirror which permits reflection of light obliquely, to light the field of the lateral telescopes. At the exit of the case the rod transmits 80 per cent of the light.

THE TELESCOPES.

The telescopes are constructed according to the same principle as those ordinarily sold, using the best available coated lenses; however, along the length of the telescope and in its metal casing is incorporated a very fine tube which will conduct air under pressure, which constitutes the anti-fogging system. The air pressure for this purpose is carried through the large flexible tube, described above, and passes through the peg which keeps the telescope in place. This is important, because the distal extremity of the telescope must be fixed in relation to the distal extremity of the quartz rod. After traveling the length of the telescope, the air is blown across the distal lens surface, thus eliminating fogging. This avoids the use of anti-vapor liquids which distort the photographic image.

The direct telescope is the most commonly used; however, to obtain good records it may be necessary to resort to various telescopes whose sighting axes vary from 30° to 120° with the axis of the direct telescope.

The special quartz for lateral lighting is indispensable. In the course of bronchoscopy it is easy to change the quartz in order to have the type best adapted to the telescope being used.

The apparatus which has just been described must be especially made, and certain of the parts have been patented by the National Center of Scientific Research. The material to be described next is in current use; its assembly alone is original. We shall consider these complementary parts and their mode of attachment to the bronchoscope.

The transformer furnished with a voltmeter and regulator adaptable for 110 or 220 volts, permits a voltage of 4-12 volts. During bronchoscopy, a voltage of four to five is used. During the photography and developing of films, it is necessary to increase the voltage to 10-11 volts. A hand or pedal rheostat makes it possible to pass from one voltage to the other.

The aspirator for drawing air through the lamp housing is one-quarter horsepower.

The compressor used for the anti-fogging flow of air is of low power.

These three parts of the apparatus together may be adjusted on an instrument table, or otherwise contained in a carrying case. In either case, a metal tube will permit the electric wires and air tubes to be grouped together, and may serve as a support for the bronchoscope. A flexible tube, about 1 meter long, and containing electric wires and the small tube of the compressor, connects the metal supporting tube to the lamp housing.

Apparatus for Cine Photography: An 8 mm. camera is used with a 12.5 mm., f. 1.9 lens, at 16 frames per second.[3] At this time an 8 mm. reflex camera permitting control while the pictures are being taken is not available, but experience shows that excellent films may be made despite this fact. A 16 mm. camera with a focal range of 17 or 20, aperture f. 1.5, 16 frames per second may also be used.[2]

The still picture photographic apparatus should be a reflex type, 24x36, focal range of 35-38 mm., aperture, 3.4. The setting should be on 0.50 m. to 0.60 m.

Camera or photographic apparatus are applied by simple contact of the camera lens to the eyepiece of the telescope, a small adapter on the camera lens will insure the alignment of the contact. This adapter will contain an additional lens, to be used with a camera that cannot be focused directly.

Optional accessories:

The Galileo Magnifier: This proximal telescope may be adapted to the bronchoscope instead of the usual bronchoscopic telescope. The most diverse bronchoscopic maneuvers are made easier by magnifying the image three times. It may be used during the introduction of the tube, the examination of the bronchi, or for obtaining specimens for laboratory examination.

The View-finder With Two Telescopes: This is composed of two Galileo view-finders, with an Eimage aspirator, permitting two simultaneous observations. It also permits observation of progress of the bronchoscopy and, therefore, is of great didactic value.

Such is the bronchoscope in its present form. To us it seems excellent. Perpetual alterations and numerous improvements to eliminate the minor disadvantages have been promised. It is essentially practical; is light, strong, but not cumbersome·

TECHNIQUE OF BRONCHOSCOPY.

We request our patients to fast the morning of the examination, to take a barbiturate on retiring the night before, and upon awakening in the morning, and we give them ½ or 1 mm. Atropine sub-cutaneously one hour before the examination. We never employ premedication with an opiate base, and only exceptionally give a curare derivative. A local anesthetic is given in classical fashion, with 5-10 cc. of tetracaine (or pentocaine) 1 per cent, with the spray and laryngeal syringe.

At the time of the bronchoscopy, the bronchoscopic tube with Jackson's light carrier may be passed through the pharynx and larynx. We prefer not to use the quartz rod lighting system during the introduction of the scope; the introduction may be difficult, and while there is some risk of breaking the quartz rod, this incident has been exceptional with us. It is important that no secretions be permitted to collect on the distal end of the bronchoscope, since such a deposit on the extremity of the rod would be difficult to remove and would prevent good light transmission; likewise before introducing the tube into the trachea or bronchi, we always check to see that no secretions are present there. It is necessary to aspirate them before the extremity of the rod or telescope becomes soiled. The success of the film depends on small details of this kind.

After these precautions, progress into the two sides of the bronchial tree is easily made. The direct telescope permits the view of a field, which is quite extensive in length and depth. It is frequently possible to descend into the terminal branches of the inferior lobe. In the course of this observation of the bronchi, it is very easy to alternate the camera and the eye, and even without a view-finder it is possible to obtain very good continuous photographs, as the scope is advanced or withdrawn, which show the character of the bronchial mucous membrane. The examination of the lateral orifices is made by changing the quartz rods and telescopes. All in all, one bronchoscopy with the taking of a film does not require any more

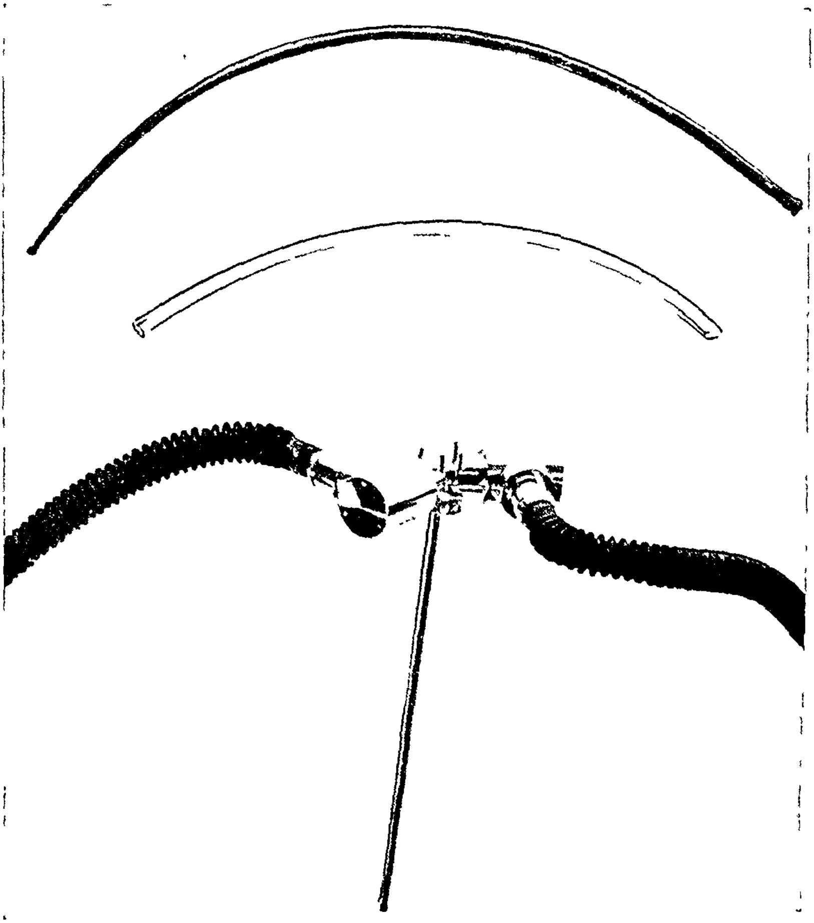


Fig. B-5. The esophagoscope: the mandril, the cover tube made of transparent plastic, the esophageal tube with double lighting.

time than an ordinary bronchoscopy; moreover, all the common endobronchic maneuvers are possible with this bronchoscope. They are performed comfortably, under very powerful lighting, which is particularly desirable if there are abundant or bloody secretions.

Cinématographic Films: 8 mm. Kodachrome "A" films at 16 frames per second are used, black and white films lacking interest. An amateur's ordinary camera is used, its only requirement being a focal range of 12.5 mm. and an aperture of 1.9. The focus is set for 0.50 m. If the camera is set for infinity, an additional lens becomes necessary. The quality of the film for clarity of image, as well as for the color quality, is really excellent. Details which were unsuspected during the bronchoscopic examination alone are evident in these films.

The bronchoscopy is completed by cinematographic study of the film on the screen. For each patient, according to the importance of the examination, one to four meters of film are taken. We believe that a film of the whole bronchial tree should be made systematically during all routine bronchoscopy. Normally, however, the photography should not prolong the examination. On the contrary the film permits it to be done in less time, since the operator knows that he can review any particular region at his leisure when the film is later projected; 8 mm. film can be enlarged to 16 mm. size for projection in large rooms, permitting the original to be kept intact. The direct use of 16 mm. films is possible, but it is intended that they be used only for teaching, or scientific publications (16 mm. Kodachrome "A" films, 16 frames per second, focal range of 17 or 20 mm., aperture of 1.5).

Finally, color slides may be made from the 8 or 16 mm. on the 24x36 mm. stock (the image then has a diameter of 10 or 15 mm.), and from these slides and enlarged color prints may be made. The diameter of the image on paper is then 75 mm.

Photography: Color photographs may be made with Ektachrome film, with a setting time of one-tenth to one-twentieth of a second, and also Kodachrome "A" at one-fourth or one-tenth degree of a second. The image on the film is from 6.5 mm. and may be projected in slide form on a screen; the en-

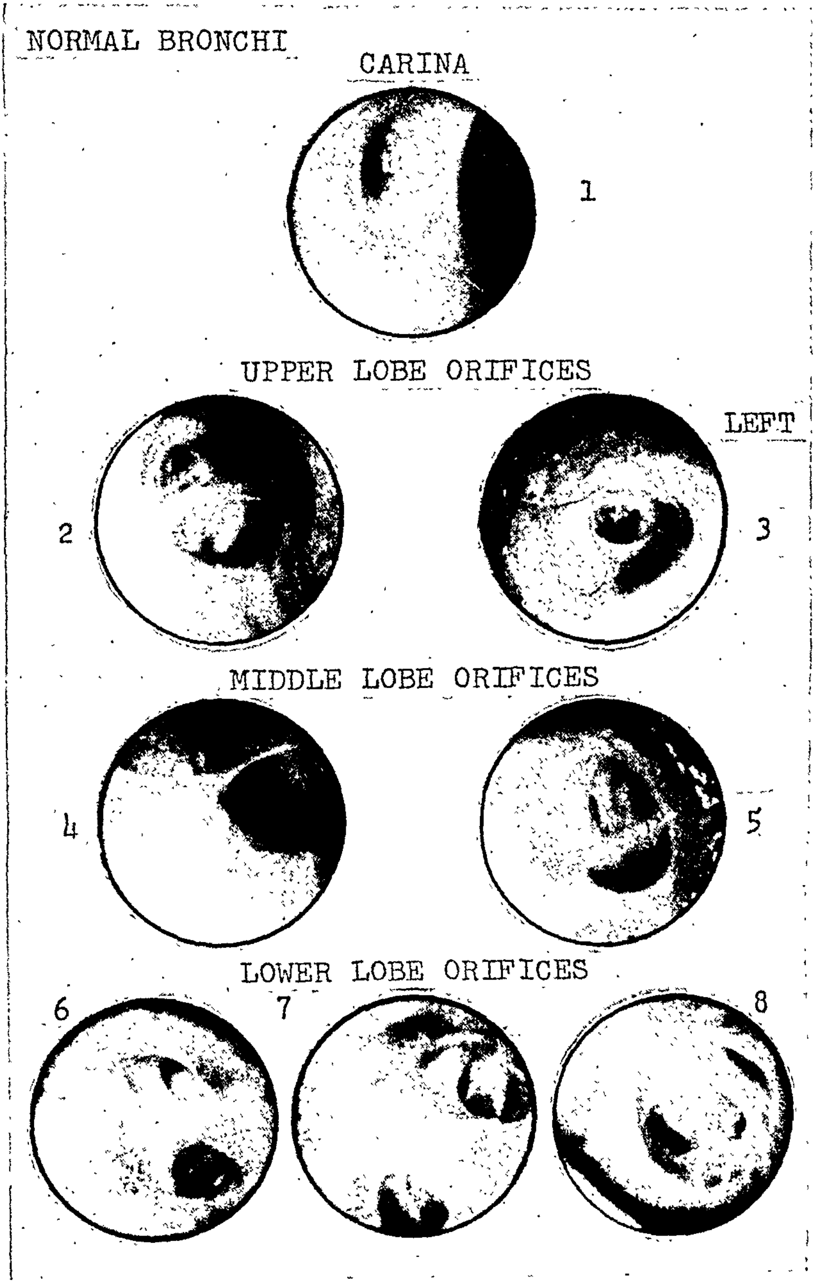

NORMAL BRONCHI
CARINA
1
UPPER LOBE ORIFICES
LEFT
2
3
MIDDLE LOBE ORIFICES
4
5
LOWER LOBE ORIFICES
6
7
8

Plate 7.

largement on paper or on film is from 24 mm. It requires the same time as does the development of cinematographic films in color, so that we make them only in exceptional cases; the cinematographic film being more economical and more interesting.

The photographs in black and white have the advantage of rapid development; they complete the filmed sequence. Ordinarily we have the print a few hours after the examination. Kodak + X films may be used, with an exposure time of one-twenty-fifth of a second. The grain is finer, and the exposure time remains sufficiently short. In all cases the image on the film has a diameter of 6.5 mm. We immediately make an enlargement on paper, which permits a perfectly clear 50 mm. image to be obtained. Enlargements as slides may also be made for projection.

The camera employed is a reflex type opening at 3.4 with a focus of 38 mm., and permitting a setting of 0.50 m. or 0.60 m.

The photographs shown here have not been retouched, intentionally; they represent those that the photographer (who has not specialized in medical photography) gives us. We be-

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Fig 1 Tracheo-bronchial spur

Fig 2 Right upper lobe orifice viewed through the right angle telescope

The apical branch is reduced to a slit visible at 3 o'clock For better examination it is necessary to use a right angle telescope The anterior orifice is visible at the top, the posterior in the middle of the field

Fig 3 A detail of the left upper lobe spur

The lingular division is clearly visible through the right angle telescope, its dividing branches may be perceived at the bottom

Fig 4 Middle lobe orifice viewed through the direct telescope

The middle lobe orifice is at 12 o'clock a secretion is perceptible at the entrance Below, the orifice of the inferior lobe bronchus

Fig 5 Orifice of the middle lobe viewed through the right angle telescope

At 6 o'clock, the departure of the lower lobe bronchus may be detected

Fig 6 Bronchi of the right lower lobe viewed through the direct telescope

At 9 o'clock, the medial basal is clearly evident.

At 5 o'clock the superior orifice with bubbling in evidence

At 3 o clock the continuation of the lower lobe bronchus where spurs of division are perceptible

Fig 7 Right lower lobe bronchi

At 5 o clock, the superior orifice is very open The first spur of the division is seen At 11 o clock the departure of the medial basal

At 2 o'clock, the termination in three branches anterior basal, lateral basal posterior basal

Fig 8 Same patient a little further the "terminal bouquet"

From the center about 3 o'clock the posterior basal, the lateral basal; the anterior basal

INFLAMMATORY ASPECTS

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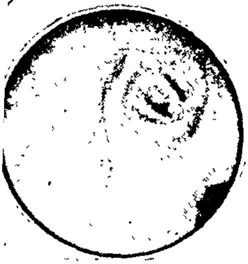

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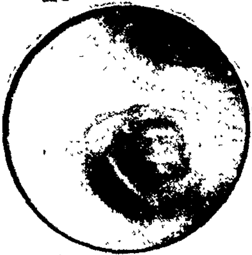

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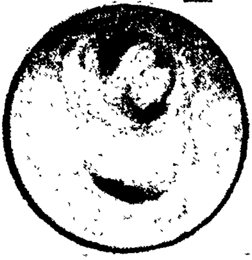

BEFORE ADRENALIN

AFTER ADRENALIN

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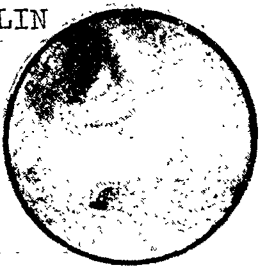

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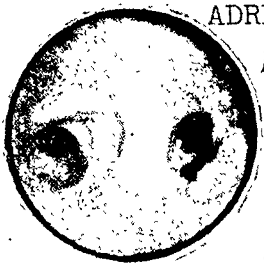

IN INSPIRATION

ASTHMA

IN EXPIRATION

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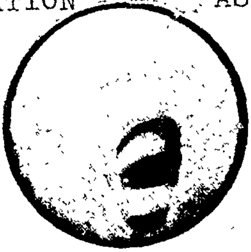

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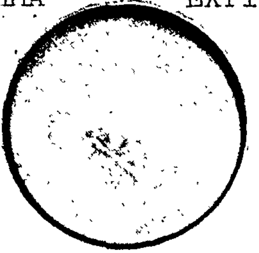
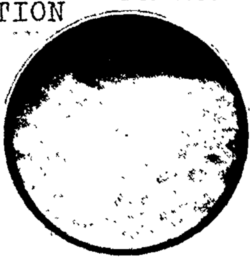


BEFORE ASPIRATION

BRONCHIAL CANCER

AFTER ASPIRATION

16



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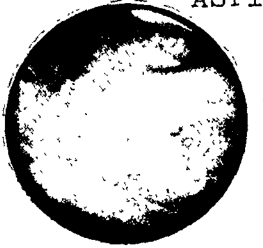

Plate 8.

lieve that the technique will be improved when a still more powerful illumination is achieved.

RECENT ACQUISITIONS AND PERSPECTIVES.

The same principle as that previously described for bron-copy, rectoscopy, cystoscopy, laporoscopy, peritoneoscopy, choscopy is applied to the whole field of endoscopy: esophagos-gastroscopy, can be performed with the same kind of apparatus. A modification of the apparatus was designed for the examination of organs which allow the use of a bigger tube, such as the rectum, the esophagus, and the stomach;[4] the lighting is double. Two light sources and two quartz rods are used. With a slight increase in the diameter of the tube, the lighting of the field of examination may be doubled (see Figs 6, 8).

With S. Segal we have published recently a special technique for esophagoscopy. We first introduce a cover tube made of transparent plastic. This is put into place in the patient, while he is sitting, with the help of a mandril; the technique is the

Fig. 9 Bronchi of the right lower lobe.

At 3 o'clock, the orifice of the superior branch; toward 12 o'clock, the medial basal; between the two, the terminal bouquet.

A certain inflammatory and edematous aspect is to be noted. The terminal branch is partially obliterated by an abnormal secretion (the left side is shown in Fig. 10).

Fig. 10. General view of the bronchi of the lower lobe.

At 11 o'clock, the left upper lobe bronchus orifice.

Toward 6 o'clock, the superior orifice; between the two, the left terminal bouquet, the classical axis of which is observed: symmetrical from the right, from outside to inside and from front to back Inflammatory aspect with purulent secretions from the terminal branches Bronchial dilatation.

Fig. 11. Detail of the left lower lobe of the same patient as in Fig. 10

The superior orifice is open widely, the termination in three branches with purulent secretions from the lateral basal.

Fig. 12 Inflammatory stenosis of the left lower lobe bronchus.

At 10 o'clock, the upper orifice and the lingula very permeable and, at about 6 o'clock, the lower lobe bronchus orifice almost entirely stenosed.

Fig 13. Same patient after local application of Adrenaline. Stenosis has partially regressed.

Figs. 14 and 15. These two figures are interesting because they clearly show the expiratory spasms in the course of asthma.

The first shows the aspect of the left lower lobe bronchus at the time of inspiration

The second shows the same region in the course of expiration. The considerable reduction of the caliber may be seen at the elimination of bronchial hypersecretions.

Fig. 16. Aspect currently encountered in a patient with bronchial cancer.

At 12 o'clock, the right middle lobe bronchus orifice; at 4 o'clock, the right lower lobe bronchus orifice from which bloody bubbles are escaping.

Fig. 17. Same aspect after aspiration

A tumoral formation of a whitish aspect is to be observed at the entrance of the right lower lobe bronchus; almost complete obliteration of this bronchus.

same as that used for introduction of a stomach tube. The patient is then put in the position of esophagoscopy. The mandril is removed and the esophagoscope can be introduced into the cover tube with security. This also avoids soiling the extremity of the tube as it passes through the mouth and throat. It is important that the cover tube be transparent, as it allows the vocal cords, the arytenoids, the opening of the esophagus and the mucus membranes of this region to be inspected as the instrument proceeds. This tube, shorter than the esophagoscope, does not impede the normal handling of the latter.

This procedure allows introduction of a rigid gastroscope into the stomach. This gastroscope can be used without difficulty for photography or cinematography.

ENDOSCOPY AND TELEVISION.

A recent experience has shown us the advantage of television.

Television can be used in the very room where the endoscopy is being performed, and a television camera can be adapted in the place of the eye or of the cinema camera. This allows everyone present in the room to follow the progress of the apparatus in the organ explored; moreover, the image, which the endoscopist and his assistants see, can be watched in the classroom of a medical school.

In this article we have explained the technique for bronchoscopy and esophagoscopy which we know very well. It is interesting to note that our technique of systematic endoscopic cinematography may be applied to all organs. In France we have specialists in rectoscopy and gastroscopy (S. Segal, M.D.), laparoscopy (Pergola, M.D.), colioscopy (Palmer, M.D.), cystoscopy (Jaupitre, M.D.), and others, and have founded an International Medical Society of Endoscopic Photography and Cinematography, with more than 300 members. The president is A. Soulas, M.D. (Paris) ; general secretary, J. M. Dubois de Montreynaud, M.D. (Reims). Dr. P. Holinger, of Chicago, and Dr Max Fourestier, of Paris, are honorary presidents.

SOME RECENT CINEMATOGRAPHIC FILMS.

1. New Technic of Bronchoscopy (systematic endobronchial cinematography), by J. M. Dubois de Montreynaud, R. J. Edwards and A J. Gladu, Reims There are four parts in this film Bronchoscopic technique, bronchial tuberculosis, bronchial cancers and asthma (experimentation in animals and observations in humans), 277 mm.

2 Esophagoscopy (personal technique, with endoscopic motion pictures, by J. M. Dubois de Montreynaud, S. Segal and G Fraisier).

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DIFFERENTIAL DIAGNOSIS OF HEARING PROBLEMS OF ADULT PATIENTS.*†

HARRIET L. HASKINS, A.M.,
Baltimore, Md.

In 1946 when Carhart analyzed the test findings of a group of patients at Deshon General Hospital, the speech hearing threshold and the average of the best thresholds in the band from 512 to 2048 c.p. showed a coefficient of correlation of about + 0.75. About one-third of all patients were found to have a difference in the two thresholds of 11 db or more. A survey of the patients over 14 years of age seen at the Hearing and Speech Center of Johns Hopkins Hospital during the past three years reveals that about 30 per cent have complex hearing problems for which routine tests give insufficient information. One suspects that there are many problems common to the subjects represented in the group Carhart statistically studied and those under audiologic evaluation at Johns Hopkins.

When routine audiologic test procedures fail to yield a consistent picture of hearing, it is necessary to inquire further into the nature of the patient's particular communication problem. The medical history, the otologic examination, the internal consistency of the battery of hearing tests and the observation of the patient's function in all communication must add up to a reasonable overall picture.

Time spent in obtaining a thoughtful, detailed medical history is often most rewarding. The patient's description of the sort of difficulty he has in communication may be very helpful. How he functions in a test situation may also be indicative of the nature of his problem. Does he arrive at threshold listening with ease and consistency? Is there an

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* Read at the Annual Meeting of the American Speech and Hearing Association held in Los Angeles, Calif., Nov. 19, 1955.

† From the Hearing and Speech Center, Department of Otolaryngology of the Johns Hopkins University School of Medicine and of the Johns Hopkins Hospital.

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indefinite range of loudness, often as much as 30 db, which might be considered threshold? Does he report that the levels where he admits hearing are really loud, and yet does not respond to slightly less intense levels? How does he function when speech sounds are comfortably loud for him? Is he confused by a moderate amount of noise when trying to understand speech? Does the patient respond to a variety of ambient noises of moderate loudness, and fail to understand speech unless he can see the speaker? Does he report that his hearing difficulty fluctuates markedly from day to day? All these behavioral factors make one question the reliability of the test results.

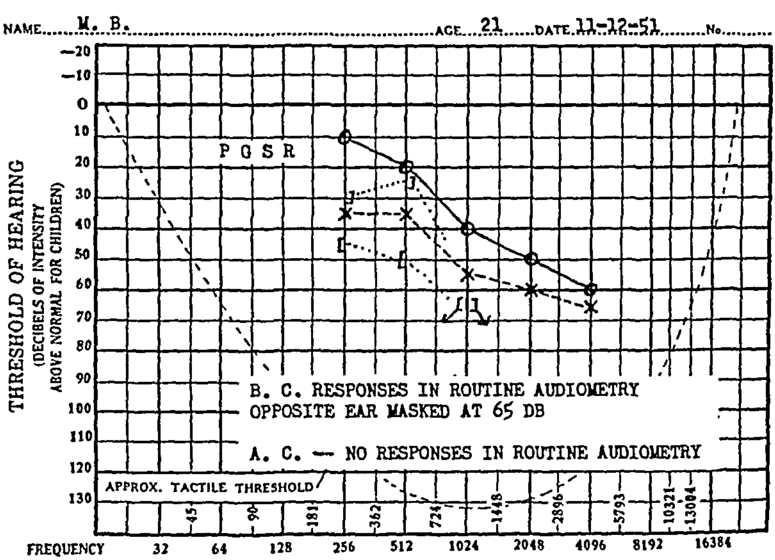
There are several other conditions which may indicate need for further study. The lack of response to all sound stimuli regardless of the intensity is legitimately very rare, for the incidence of total deafness is negligible. The person who has a mild to moderate amount of difficulty in communicating and has indicated threshold hearing at levels of 70-80 db has not given an accurate test picture. Patients who have extreme difficulty in understanding speech and yet repeatedly report that they hear the sound of the voice at quite low levels are in need of further study. It is found that their communication does not improve, either by the use of comfortably loud voice or amplification; a hearing aid apparently creates confusion of noise in the ear without promoting clarity. Then, too, the person who achieves a gain of 60-75 db with a hearing aid needs further investigation.

Three of the most useful measurements of hearing function are:

1. Threshold readings for a pure tone spread of at least 6 octaves.
2. The relationship between the responses to air conduction and bone conduction stimuli.
3. A test of ability to hear fine differences among speech sounds.

The third test, known as the test of discrimination, has proved the most useful of all. It is the best single index available for determining how effectively one can use his hearing. When the ability t speech sounds is markedly impaired, it is important to attempt to find the cause.

The use of psychogalvanic skin resistance audiometry provides pertinent information about the function of the end-organ of hearing. This test by no means gives all of the facts which one should like in some complex cases, but it does give one more measurement that is free from various behavioral complications. Such additional information often makes possible a more accurate prognosis for the patient and aids in planning an appropriate course of therapy.



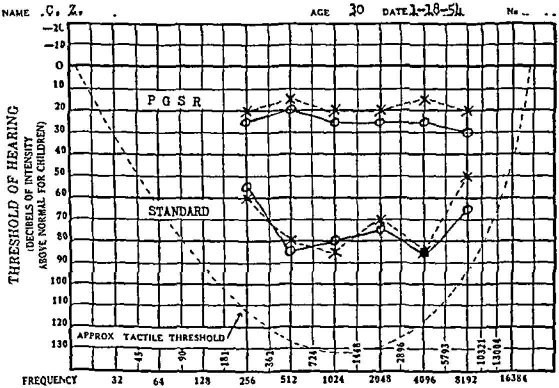
SPEECH AUDIOMETRY

FIRST AWARENESS	COULD NOT DO
"HEARING THRESHOLD"	COULD NOT DO

Fig. 1.

It is worthwhile to consider the records of a group of patients for whom the results of a test by psychogalvanic skin resistance audiometry provide a critical measurement of hearing function and help to clarify the interpretation of the overall picture. Some of the problems found among the group considered here are those with a history of meningitis, lues, psychological disorders, familial hearing impairment, conditions that appear to be the result of vascular sclerotic changes, long-term severe impairment and auditory perceptive and language disorders.

The first (Fig. 1) is a 21-year-old girl who suffered a severe, undiagnosed illness at the age of 12. A form of encephalitis or meningitis was suspected. She had a fever of 104° for several days and was unconscious for periods during this time. On the tenth day of this illness, tinnitus developed, and she lost her hearing. She was able to finish high school with honors. On examination, one notes marked changes in her voice quality. She appears to be an unusually good lipreader. She reports that she does not hear any sounds regardless of the intensity level. There were no subjective responses to pure tone stimuli by air conduction. There was a subjective response to bone conduction stimuli at 512 cp during the initial test only. The measurement obtained by psychogalvanic skin resistance audiometry indicates a moder-



SPEECH AUDIOMETRY

FIRST AWARENESS 34 DB

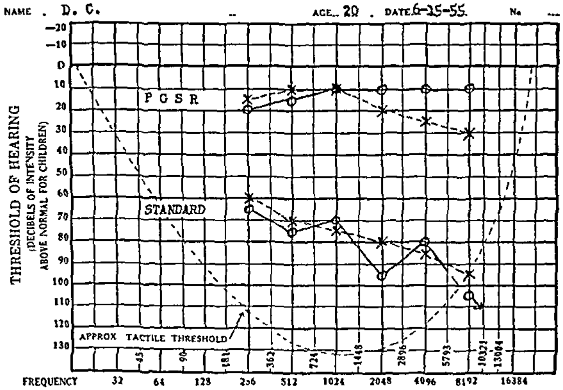
"HEARING THRESHOLD" COULD NOT DO

Fig 2

ately severe bilateral impairment of the end organ of hearing; however, one could not be sure that this problem was not further *complicated by a disorder of auditory perception.* Because of this question and other manifestations of behavioral disorders, the patient was referred to a psychiatrist. He observed that, under hypnosis, she could understand a conversational voice of moderate intensity by audition alone. It is clear that this problem involves both a moderate organic impairment and a functional disorder.

Fig. 2 shows the audiometric findings for a 30-year-old man who had been treated periodically for 13 years for lues. There were no other pertinent medical findings. He reports that he has had difficulty hearing for about eight years. He has tinnitus and reports that at times his hearing fluctuates. He attempts to use a hearing aid sporadically but cannot understand speech by auditory cues with or without the aid. Sometimes mild amplification creates only noise in his ear. The patient's speech is good, and his language understanding by visual cues and language usage are also good. Note the levels of response reported by routine audiometric procedures. These responses were inconsistent, and it was difficult to determine a threshold measurement. On three different occasions the patient indicated an awareness of the sound of the voice at about 30-34 db. This is a less complicated listening situation for the patient than those of pure tone and speech hearing audiometry. The test by psychogalvanic skin resistance audiometry reveals that the patient has no worse than a mild impairment in the function of the end organ of hearing. His problem is severely complicated by difficulty in auditory perception. Hearing aid use is of minimal benefit to this patient and he must continue to rely on visual cues.

Fig. 3 is a 20-year-old boy who was first examined while attending a school for the deaf. He is reported to have congenital deafness. His mother and her brother have like hearing problems. This boy responds to a great variety of ambient noises but does not understand speech by auditory cues. His own speech is limited, but it is articulated within a normal time span. He enjoys using a mild gain hearing aid. His language understanding by lipreading and his speech have improved noticeably since he left school and has spent most of his time with hearing people, and has had lessons in speech and language work. Note the first awareness response level of 16 db above normal and the audiogram by psychogalvanic *skin resistance of near normal level. This does not appear* to be a problem of impairment of the end-organ of hearing, but one of auditory perception and/or language association.



SPEECH AUDIOMETRY

FIRST AWARENESS 16 DB

"HEARING THRESHOLD" COULD NOT DO

Fig 3

The fourth case (Fig. 4), is a 53-year-old woman who is a personnel counselor in a large high school. When first seen she was in poor physical condition and worried about her rapidly progressive hearing impairment, which she feared would soon incapacitate her for her position. She has a positive family history of "progressive deafness" and had anticipated serious problems herself. Her father was deaf for many years and was a very poor communicator. This patient was attempting to use a powerful hearing aid when we first saw her. The noise in the instrument and the amplified sound about her were very distracting. She had ceased to mingle socially and often stayed away from work because it was an exhausting experience. Note the awareness level of 26 db which is consistent with the audiogram made by psychogalvanic skin resistance method; also, the great variance in her discrimination scores. This patient has a moderate bilateral nerve type hearing impairment. After medical treatment her general condition was improved. She was assured of the relatively moderate degree of her impairment and advised that any progressive aspect of her hearing loss could not be determined. She was fitted with an instrument appropriate to her degree of loss and counselled in its use. A more recent test shows a speech hearing threshold of 44 db instead of the original 50-62 db, and with her new aid she achieves a threshold of 14 db. and a mild loss of 12 per cent in discrimination. This is reasonably good function for this degree of end-organ impairment.

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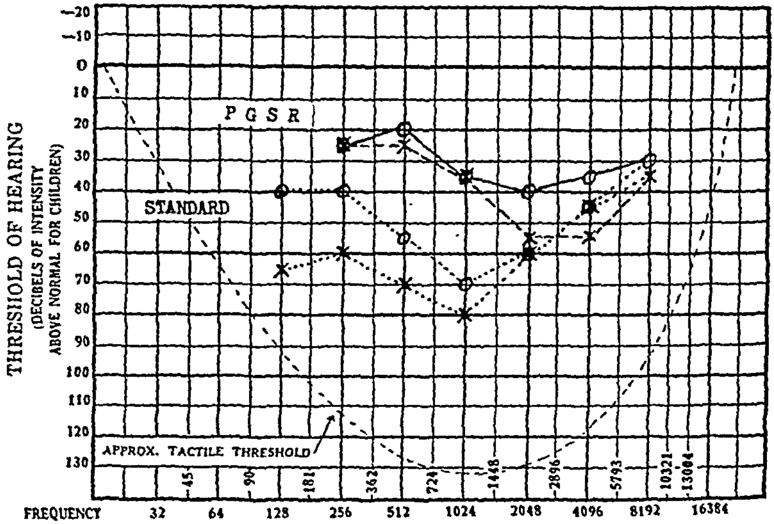
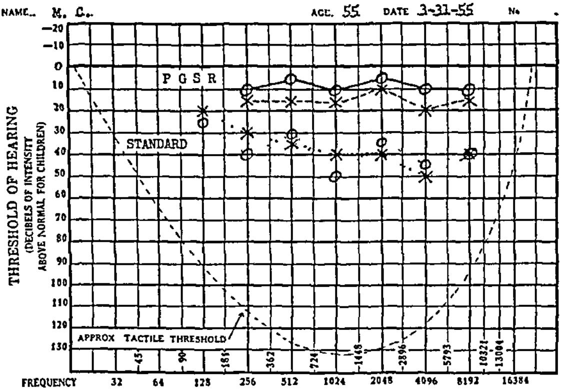


SPEECH AUDIOMETRY		DISCRIMINATION TESTS * % LOSS	
FIRST AWARENESS	26 DB	AT SLOW RATE IN QUIET	56
"HEARING THRESHOLD"	50-62 DB	AT NORMAL RATE IN QUIET	26-68
		AT NORMAL RATE IN NOISE	—

Fig. 4.

Fig. 5 refers to a 55-year-old woman who has had difficulty hearing for three years. The otologic examination is negative. There is no history of ear infection nor of a familial hearing impairment. She does have tinnitus. She communicates fairly well in an easy listening situation, when the background noise is minimal and one person nearby is speaking to her in a moderately loud voice and at an even rate. Attempts to use a hearing aid have only complicated her problem Note the



SPEECH AUDIOMETRY		DISCRIMINATION TESTS * % LOSS	
FIRST AWARENESS	26 DB	AT SLOW RATE IN QUIET	14
"HEARING THRESHOLD"	46 DB	AT NORMAL RATE IN QUIET	28
		AT NORMAL RATE IN NOISE	56

Fig 5

near normal thresholds by psychogalvanic skin resistance audiometry; also, that awareness for sound is 20 db. better than the speech hearing threshold. A difference of 6 to 10 db is usually observed in subjects whose audiometric test is relatively flat Note the marked loss in discrimination of 28 per cent when test items were presented at normal rate in quiet. This seems to be a case of phonemic regression. The end-organ of hearing is functioning within range of normal limits. It appears that in this case the difficulty in understanding speech is probably caused by changes in the auditory perceptive mechanism.

The use of psychogalvanic skin resistance audiometry is a special and exacting technique used in the diagnostic process at a medical center. It appears to test the function of the end-organ of hearing. It provides a measurement which assists in differentiating between the receptive hearing function and the combined function of receptive and perceptive hearing mechanisms. This information is invaluable to the otologist and audiologist in understanding the patient's problem and in planning a feasible course of therapy.

ANNOUNCEMENT.

The Mount Sinai Hospital, New York, in affiliation with Columbia University announces an intensive postgraduate course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty given by Irving B. Goldman, M.D., and staff, July 14, 1956, to July 28, 1956. Candidates should apply to Registrar for Postgraduate Medical Instruction, The Mount Sinai Hospital, 5th Avenue and 100th Street, New York 29, New York.

UNIVERSITY OF ILLINOIS, COLLEGE OF MEDICINE.

The next Laryngology and Bronchoesophagology Course to be given by the University of Illinois, College of Medicine, is scheduled for the period November 5 through November 17, 1956. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois, College of Medicine, 1853 W. Polk Street, Chicago 12, Ill.

THE FUNCTION OF A MEDICAL SOCIETY SPONSORED HEARING CENTER.*

HAYES A. NEWBY, Ph.D. (By invitation),

Palo Alto, Calif.

We specialists in the field of audiology feel a very close and warm relationship with you who practice the medical specialty of ear, nose and throat. We like to feel that we are all working together in the dedication of our efforts for the betterment of individuals with impaired hearing. I have been asked to speak on the topic of "The Function of a Medical Society Sponsored Hearing Center." Before getting into this specific topic, however, I feel it would be worthwhile to spend some time reviewing the development of this new specialty of audiology and the growth of the community hearing and speech center.

Fifteen years ago there were no hearing and speech centers, and the word "audiology" was unheard of. The first "hearing and speech centers" were the military aural rehabilitation centers of World War II days, where, for the first time, the combined abilities of various specialists were concentrated on the diagnosis and treatment of hearing impairment. In these military centers the meaning of the words "diagnosis" and "treatment" was extended far beyond their usual medical connotation.

In the extension of the meanin
ment" lies the definition of clini
concerned with "diagnosis" from
the hearing function of an indivi
cess of surgical procedures or pros
his hearing loss. Audiology is conc

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* Read at the meeting of the Western [illegible] Rhinological and Otological Society, [illegible]

Editor's Note: This ms. received [illegible] for publication, March 26, 1956.

the sense of providing rehabilitation in the form of auditory training, speechreading, and speech training for those who have permanent impairment of their hearing function.

Much has been written and said about the lineage of this new field of audiology. While it is true that its ancestors include various medical specialties, psychology, physics and education, audiology is a direct descendant of the fields of speech correction and otology. The other fields I mentioned may be "kissing cousins" of audiology, but its "mammy" and "pappy" are speech correction and otology. Sometimes audiology dwells with the one parent and sometimes with the other. For example, at the University of Pittsburgh audiology is a division of the Department of Otolaryngology in the medical school, while at Northwestern University audiology is part of the Department of Speech. Sometimes audiology leaves the parental home to live alone, though with close ties both to mother and father; for example, the Portland Center for Hearing and Speech, the Seattle Hearing and Speech Center, and the San Francisco Hearing and Speech Center. It is with these community audiology centers, rather than the university sponsored centers, that this paper is primarily concerned.

To carry the analogy further, audiology is the product of a "marriage of convenience." During the war the medical departments of the Army and Navy asked for assistance in setting up programs of aural rehabilitation. For this assistance medicine turned to university speech clinics and recruited specialists in speech correction, experimental phonetics, and in the education of the deaf. Together with otologists these non-medical specialists devised tests for measuring the social efficiency of hard-of-hearing individuals; tests for malingering and other types of functional deafness; tests for the selection of hearing aids; and they planned a program of rehabilitation for those who were handicapped because of their hearing impairment. The success of the combined efforts of the medical and non-medical specialists was measured in the excellent record established by the armed forces in returning hard-of-hearing and deafened service men to duty, or returning them to civilian life with a minimum of handicap. Neither group of specialists could have accomplished this job single-handedly; together they created the field of audiology.

You are all familiar with the post-war development of audiology and the mushrooming of audiology clinics, or hearing centers, which today have become hearing and speech—or speech and hearing—centers. The techniques of assessment of hearing function, selection of hearing aids, and rehabilitation of the hearing-handicapped which were developed during the war have been improved and extended in these civilian centers. Perhaps the outstanding post-war innovation in audiology has been the focusing of attention on the training of pre-school deaf and hard-of-hearing children and their parents. In most centers, children make up a considerable proportion of the case load.

During the war, audiology programs were under the direction of medical officers according to the military table of organization. When the war was over the otologists who had been concerned with aural rehabilitation programs returned to their civilian practice. Many of these otologists, being well aware of the accomplishments of the military audiology program, desired to see a similar program established for the civilian population. Thus most hearing and speech centers today owe their existence to the pioneering efforts of one or more otologists.

The administrative direction of community audiological programs has largely passed from the otologist's hands, however. This is a natural development, since there are few otologists whose sole interest is in audiology or who can afford to devote the major share of their efforts to the administration of audiology programs. During the war it was different; then otologists were ordered to duty, and they had no private practice. Post-war audiology programs were usually started on a financial shoestring and depended on public support for their continued existence. The direction of such an audiology program required more time than most otologists could give; so, while otologists were largely responsible for the creation of community audiology centers, they were forced by circumstance to relinquish administrative control to non-medical specialists, and since the war the development of community audiology centers has been primarily along non-medical lines.

Just as the interest of otologists in the field of audiology was stimulated by their participation in the military aural rehabilitation programs, so also was the interest of the non-medical specialists who, together with the otologists, had pioneered in the military centers. These non-medical specialists, most of whom had been recruited from college speech clinics, realized that audiology offered them new opportunities for service for which their previous training and experience had to some extent prepared them. Some of these non-medical specialists returned to their universities to organize and direct training programs in audiology, and some became the administrative directors of community hearing and speech centers. What before the war had been university speech clinics, shortly after the war became speech and *hearing* clinics. The impact of the growth of audiology along non-medical lines is nowhere evidenced more forcefully than in the reorganization in 1947 of the American Speech Correction Association into the American Speech and Hearing Association, and the change in the title of that organization's official publication from the *Journal of Speech Disorders* to the *Journal of Speech and Hearing Disorders*. By 1950, extensive graduate training programs leading to the M.A. and Ph.D. degrees in audiology, or in speech and hearing, were in existence in a dozen of the leading American universities. The products of these training programs were ready, willing and able to assume clinical and administrative responsibilities in community hearing and speech centers. The field of audiology had "come of age."

Most otologists have accepted in good grace this development of audiology as a non-medical specialty and have been content to assume an advisory capacity to audiology centers in medical matters, welcoming the extension of audiological services to the general public which the centers have made possible. There are some otologists, however, who have played the role of the over-protective parent who hates to see his child grow up and assume independence. Such otologists deprecate the contributions of the centers which they helped to found and look down their noses at the non-medical clinicians who perform the day-to-day work at these centers. Some feel that in helping to develop the specialty of audiology they have created a Frankenstein monster which is now completely out of control. Fortunately, these deprecators and complainers are in the minority; the great majority of otologists are playing the role of well-adjusted and proud parents who have accepted audiology as a trusted and valued professional partner.

In all fairness to the complainers it must be admitted that some audiology programs have developed improperly and unprofessionally. In the creation of a new field it was inevitable that there would be mistakes. Some community centers were brought into existence by overly-ambitious individuals who were more interested in expansion for expansion's sake than in sound professional growth. In some centers there was far too little medical supervision; but by and large, hearing and speech centers around the country have developed soundly and with excellent medical guidance.

So much by way of introduction to the specific title of my paper. I want now to tell you how one community audiology center was created and how it has developed. I believe that the San Francisco Hearing and Speech Center, of which I am Director, has the unique distinction of being the only audiology center founded by a medical society. In 1948 the San Francisco Center was created through the joint efforts of the San Francisco Medical Society and the San Francisco Hearing Society. Professional guidance of the Center was provided by a committee of six otologists appointed by the President of the Medical Society. Originally, the Director of the Center was responsible solely to the committee of otologists. A little less than a year ago the Center was chartered as a non-profit corporation. The administrative control is now vested in a board of directors on which otologists are represented. The President of the Medical Society continues to appoint a Hearing and Speech Center Committee, which is now composed of four otologists and a pediatrician. This committee serves in an advisory capacity to the board on medical matters pertaining to the Center.

The function of the San Francisco Hearing and Speech Center is to provide specialized audiological services for referring physicians and medical agencies. One of the services offered by the Center is diagnostic audiometry, which means the assessment of the hearing function as an aid to the otological diagnosis of a hearing impairment. Diagnostic audiometry now includes also the examination of very young children who are suspected of having hearing impairment because of their failure to develop oral language. A second service is assessment of the hearing function in individuals who are candidates for the fenestration or the stapes mobilization operations, and the evaluation of the results obtained through these surgical techniques. A third service is hearing aid consultation—the advising of patients primarily as to whether they would be benefited by using any hearing aid, and secondly, making recommendations of a more specific nature in regard to hearing aids. In the fourth place, the Center provides rehabilitative services on an individual or group basis for children and adults with permanently impaired hearing. Finally, the Center operates a training program for pre-school deaf youngsters and their parents.

At present the Center is serving a case load of approximately 1200 patients a year. No patient is accepted without a medical referral, and 60 per cent of our case load comes on referral from private physicians. In the course of a year referrals will be received from as many as 125 different physicians in the Bay Area. Twenty-five per cent of the case load is referred from various governmental agencies, such as the Bureau of Crippled Children Services of the State Department of Health, the Bureau of Vocational Rehabilitation, and the Welfare Department. The remaining 15 per cent of patients are referred through other medical agencies, primarily ear, nose and throat clinics in various hospitals in the Bay Area. In every case, reports of the Center's examinations and recommendations are made to the referring physician or agency, so that there is close liaison between the Center and the medical referring source.

In 1948 the Center's staff consisted of one audiologist who performed his own secretarial work. From this modest beginning, the Center has developed a staff of eleven people, consisting of a Director, an Associate Director, a Research Director who doubles as a clinical audiologist, four other clinical audiologists, a test assistant, a nursery school teacher who works also part-time as a bookkeeper; an office manager, and a stenographer. At the San Francisco Center there is no resident otologist. In addition to the physicians on the medical advisory committee who are available for consultation, there is a pediatrician who is appointed by the President of the Pediatrics Section of the Medical Society to serve as medical advisor to the pre-school training program.

Like most activities of its kind, the San Francisco Hearing and Speech Center has been beset by financial problems from the time of its founding. Rehabilitation is an expensive business and can never be self-supporting if services are to be available to all who need them. The San Francisco Center obtains only one-third of its support from patient fees. The rest of its budget comes from civic-minded organizations and individuals who have appreciated the value of the Center's contribution to the hearing-handicapped. Prominent among its supporters have been the Junior League of San Francisco, the Rotary Club of San Francisco, Infant Shelter, the San Francisco Hearing Society, and Mount Zion Hospital, where the Center is presently housed. Financial support has also been received from various foundations and governmental agencies. Most hearing and speech centers receive Community Chest support. To date, the San Francisco Center has not been affiliated with the Chest, although it is contemplated that application for Chest membership may soon be made.

The description of the San Francisco Hearing and Speech Center's activities would not be complete without paying tribute to the otologists who have been instrumental in its founding and development. I am pleased at this opportunity to acknowledge our debt of gratitude and continuing appreciation to Drs. Robert C. McNaught, Robert C. Martin, Walter P. Work, Shirley H. Baron, and William T. Duggan.

In many respects the history and development of the San Francisco Hearing and Speech Center parallel the history and development of hearing and speech centers in all parts of our country. The growth of the audiology center as a community responsibility and activity stems from the interest of otologists whose visions of a civilian aural rehabilitation program originated from their experience in the military service. In addition, of course, the development of the community center is due to the interest and vision of non-medical personnel whose basic training for the most part was obtained in the field of speech correction. Thus audiology is the child of parents of somewhat different background. I feel that both parents can look with pride on the accomplishments of their precocious child, and I believe that the future of audiology lies with its continuing allegiance to both parents. I hope that audiology will always obey the Biblical injunction to "Honor thy father and thy mother."

REVIEW OF THE CORTICOSTEROIDS—THEIR APPLICATION TO OTOLARYNGOLOGY.*†

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Hydrocortisone is the most important steroid secreted by the adrenal cortex. Like cortisone and the newer synthetic compounds (prednisone and prednisolone), it is a so-called gluco-corticoid and has only slight mineral and water retaining activity. In the remarks to follow, the terms corticoids, corticosteroids, and gluco-corticoids will be used interchangeably to designate hydrocortisone or any of the cortisone-like hormones.

Approximately 100 mm. of hydrocortisone are secreted in 24 hours by the intact, normal, human adult. The main function of this hormone is the preservation of healthy tissue and healthy cells, not only in normal physiologic conditions, but also during stress of any kind. Stressed tissues send impulses to the hypothalamus, which in turn stimulates the anterior pituitary to secrete ACTH. The latter stimulates the adrenal cortex to secrete hydrocortisone. Increased secretion of ACTH also results when the blood level of hydrocortisone is low. A third stimulus to increase the secretion of corticotropin from the anterior pituitary is the direct action of adrenalin, the hormone from the adrenal medulla.

In this function of maintaining homeostasis, hydrocortisone may produce many physiologic actions. I have reviewed these actions in a recent paper[1] and therefore, will not repeat them now. Instead, an attempt will be made to elaborate on only three actions of the corticosteroids which seem especially important in the field of otolaryngology. These three actions are: *1.* anti-inflammatory action; *2.* action in regard to infection, and *3.* action on capillary and connective tissue permeability.

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† From the Department of Otolaryngology, Stanford University School of Medicine.

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Before reviewing these three subjects, a short resume on the anatomy and physiology of the connective tissue seems in order. Loose connective tissue, such as that found under the epithelium of the nose, sinuses and most of the respiratory tract, consists of a homogenous gel-like matrix in which are inmeshed a mass of fibrils and fibers, cells, lymphatics and capillaries. We must not forget that the capillary bed is a part of the connective tissue. Another important fact, which was recently emphasized by Dougherty,[2] is that no capillary or blood vessel supplies nourishment directly to any cell in the body. Passage through the ground substance of the connective tissue is essential before the cells of any organ are reached by anything from the circulation; hence, the circulation in the ground substance is almost as important as that in the vascular bed. The most important cellular elements of the connective tissue are the fibroblasts and mast cells.

The mast cells are large granular basophilic staining mesenchymal cells, which, according to Asboe-Hansen,[3] function in forming the ground substance. The granules of these cells contain histamine, and also a heparin-like mucopolysaccharide which is a hyaluronidase inhibitor. Stress of any kind causes the mast cells to "spit" their granules into the gel of the connective tissue, thus liberating these substances. The corticosteroids also degranulate these cells. The mast cells have recently taken on great importance in regard to connective tissue, and undoubtedly are essential in maintaining the integrity of the ground substance. The connective tissue under the epithelium, lining the nasal membranes, contains many mast cells. The fibroblasts are more concerned in the forming of the fibrous elements of the connective tissue. The ground substance is normally a gel and is composed of protein and carbohydrate molecules. These molecules are polymerized (aggregated) to form mucopolysaccharides.

The most important of the polysaccharides is hyaluronic acid. The entire connective tissue is metabolically active and, hence, is in a continual state of flux. In other words, it is being constantly built up and constantly broken down. Enzyme action accounts for most of this activity. For example, the ground substance is built up by enzymes from the mast cells and, possibly the fibroblasts, and is broken down, liquefied or depolymerized by the enzyme hyaluronidase. Thus, hyaluronidase changes the ground substance from a normal gel to a more or less of a sol. While hyaluronidase, in any important amount, has not been found in ground substance, Mayer[4] has shown that in inflammation there is a forty fold increase in this enzyme; hence, its precursor must be present in the connective tissue at all times. The consistency of the ground substance is of great importance to our physiology and our normal defenses. As stated previously, nourishment, carbon dioxide and oxygen, as well as metabolites and substances secreted by parenchymatous organs, must go through the ground substance before reaching their destination; also, invading bacteria and viruses, after passing through the epithelium, must go through the ground substance before reaching the blood stream or before reaching deeper important structures. For example, a virus which gets through the nasopharyngeal epithelium, must penetrate the ground substance before getting into the vascular bed; therefore, if the gel is firm, it provides a good barrier against invasion of these infecting organisms. A more liquid edematous ground substance allows a better chance for invasion. Some of the important streptococci, pneumococci and staphylococci produce hyaluronidase themselves and, hence, are particularly invasive. Other points about the connective tissue will be pursued further in the observations to follow.

THE ANTI-INFLAMMATORY ACTION OF THE CORTICOSTEROIDS.

One of the most important, interesting and incompletely understood actions of the glucocorticoids is their anti-inflammatory action. Due to this action, Selye[5] has named this group of corticosteroids *antiphlogistic hormones*. This is in contradistinction to the phlogistic hormones such as thyrotropin and growth hormone.

Dougherty[2] has brought out the fact that inflammation always occurs in the connective tissue. This very important fact has not been generally recognized. When one peers into an inflamed nose, whether the inflammation is of allergic, infectious or traumatic nature, the swelling is not in the epithelium, but is in the edematous ground substance of the connective tissue under the epithelium. In inflammation, we get an increase in the connective tissue cellular elements, along with an increase in leukocytes and other wandering cells; also, the ground substance becomes edematous, and the capillaries engorged. The corticosteroids depress all of these processes.

Now, what is inflammation? It is a chain reaction[6] in the connective tissue, which is the result of stress. The stress produces cell damage which liberates certain substances which set off and continue the inflammatory reaction. The damaged cells are mainly fibroblasts and mast cells. Not all of the released substances are known, but among them are histamine, hyaluronidase, heparin-like mucopolysaccharides and leucotaxin. Thus, local inflammation and generalized stress differ only in degree and localization of the stress.

Dougherty[6] gives the following hypotheses as possible mechanisms in corticoid anti-inflammatory action:

1. Decrease in the level of circulating fibrinogen, thus producing a decrease in the amount of fibrin at the site of inflammation.

2. Inhibition of hyaluronidase, thus decreasing the action of the spreading factor.

3. Inhibition of the formation of hyaluronic acid of the ground substance.

4. Shedding of mast cell granules with release of a heparin-like mucopolysaccharide, which has anti-hyaluronidase action and which also binds histamine.

To this list, could be added the action of maintaining a good capillary bed. Dougherty[6] believes that all of these actions take place, but that there may be a more fundamental anti-inflammatory phenomenon involving the action of hormones on the many substances liberated from cell damage.

INFECTION AND CORTICOSTEROIDS.

There is no question but that the gluco-corticoids in *large* doses impair the host's resistance to many infections. There are numerous explanations for this. Thomas[7] states that there are at least six possibilities. They are as follows:

1. A Direct Action on the Infecting Organism. There is no direct evidence that this takes place.

2. An Effect on the Neutrophils. Although the corticosteroids produce a systemic leukocytosis, fewer pus cells migrate to the area of local inflammation. There is slight evidence, but no definite proof, that the bactericidal properties of leukocytes are diminished. Some virus infections are also enhanced by *large* doses of corticoids and since available evidence shows that neutrophils are not important in virus infections, the leukocyte hypothesis becomes somewhat untenable.

3. Influence in Antibody Formation. There is no definite evidence that this is a factor in humans.

4. Interference With the Inflammatory Reaction. The corticoid depression of tissue inflammation is an accepted fact, but as noted above, the explanation is uncertain. At any rate, the anti-inflammatory theory does not explain the enhancement of virus infections, nor the enhancement of bacteria which are injected directly into the blood stream. In these two instances, there is no local inflammatory reaction.

5. The effect on serological mechanisms, such as the nonspecific hyaluronidase inhibitor.

6. Damage to the Reticuloendothelial System. The macrophages in this system are for the purpose of clearing the blood stream of bacteria and particulate matter. There is some evidence that like certain colloids, the corticoids block the cells of the reticuloendothelial system so that these macrophages then cannot clear the blood stream of infectious organisms.

It can readily be seen that the phenomenon whereby *large* doses of corticoids enhance infection, is not easily explained by any one hypothesis.

On the other side of the picture, Robinson,[8,9] et al., Vollmer[10] and Lurie,[11] et al., give evidence to show that in *small* doses, these hormones not only fail to enhance infection, but also aid the infected host and protect the host from infection. Robinson states that to date many patients have received cortisone, and the number of reports citing intercurrent infection has been negligible.

Jawetz[12] states that in patients seriously ill with infection and toxemia, the antitoxic effect of cortisone may save the patient while a proper antibiotic is destroying the infection.

It would appear that, as far as infection is concerned, with doses of corticoids large enough to produce symptoms of hyperadrenocorticoidism, there is enhancement of the infection; and that with very small doses, or with short term dosage, there may be improvement of the host with inhibition of the infection.

I have used this fact, probably advantageously, by giving small doses of hydrocortisone or ACTH for a short time in the early stages of virus upper respiratory infections. The combination of ascorbic acid and one of the bioflavonoids, also firms the connective tissue gel, by antihyaluronidase action,[13] and hence, can be used with corticoids in this connection.

CORTICOSTEROIDS IN RELATION TO CAPILLARY PERMEABILITY AND CONNECTIVE TISSUE PERMEABILITY.

The work of Zweifach[14,15,16] has shown that the exchange of substances between the blood stream and parenchymatous tissues or epithelial structures is a much more complicated matter than had previously been described. In order better to visualize it, some of the anatomy and physiology of the peripheral vascular bed will be described.

In the first place, the capillary endothelium and the inter-endothelial cement substance are not the only structures involved in this matter. There are at least five structures involved, and they are grouped together under the term hematoparenchymal barrier. They are as follows:

1. *A Thin, Slimy Proteinaceous Substance Lining the Inside of the Capillaries.* This apparently comes from the blood plasma. Zweifach's experiments show that if it is washed away, some edema will occur.

2. *The Endothelial Cells.* While water and large molecules pass through the inter-endothelial cement, carbon dioxide, oxygen and lipid soluble substances diffuse mainly through the endothelial cells; hence, the maintenance of healthy endothelial cells, which the corticoids aid in doing, is very important in providing nourishment and oxygen to the cells of the important structures in our specialty.

3. Inter-endothelial Cement. This substance is probably a calcium proteinase and is secreted continuously by the endothelial cells. Endothelial cement makes up only a very small amount of the total capillary surface (about one per cent), but is important in the passage of water and large molecules. Proteolytic enzymes will break down the cement substance, but hyaluronidase will not. The pH of blood markedly affects the permeability of this structure, and, adequate calcium is necessary in order to maintain its normal consistency; however, Zweifach has found that hypoxia does not increase its permeability. It was formerly thought that practically all substances passed through the cement substance, but as noted previously, oxygen, carbon dioxide and lipid soluble substances pass mostly through the endothelial cells.

4. Peri-capillary Sheath. This structure is of the utmost importance, particularly in relation to edema. It is a condensation of fibrils and very firm ground substance which supports the capillary. When it is firm, it prevents edema. When it is liquefied by hyaluronidase, edema occurs. The corticoids help to keep this structure firm and prevent edema. Ascorbic acid and the bioflavonoids also aid in this regard.

5. The Connective Tissue. Like the peri-capillary sheath, the consistency of the connective tissue ground substance is also affected by the corticoids.

It can be seen that in the production of edema, several of these structures may be involved; also, all of these structures are in a constant state of metabolic flux and vary considerably in their physical and chemical make-up. At times they are more permeable than others. Of special importance is the pericapillary sheath. This structure exerts the ultimate control of the capillary permeability and the latter's permeability characteristics are regulated largely by the corticosteroids.

There is another factor about edema which has nothing to do with capillary permeability. This factor is that of the action of hyaluronidase on the ground substance. Since hyaluronidase is liberated in inflammations, it liquefies the ground. substance and, thus, produces edema directly. This is probably an important factor. Since corticosteroids are anti-hyaluronidase factors, their action helps prevent edema in this manner; thus, the production of edema, whether it be from allergy, infection or trauma, is a complicated matter and much more complex than simple osmosis and hydrostatic pressure.

The contraindications to the use of the corticoids and ACTH are too well known to dwell on them at this time; however, I emphasize the fact that large doses of these hormones over long periods of time will depress the adrenal cortex. To me, this is the most dangerous factor in their use; therefore, in otolaryngologic practice, whenever possible, we should not resort to this type of therapy. In surgical cases, however, if there is any possibility of cortical depression from previous therapy, prophylactic corticoids should be given before and for a few days following surgical stress; also, intravenous hydrocortisone should be available for emergencies.

OTOLARYNGOLOGIC CONDITIONS IN WHICH THE CORTICOSTEROIDS AND ACTH ARE USEFUL.

1. Hay Fever.

Particularly in severe cases not controlled by ordinary methods and in localities where the season is short. Besides systemic therapy the local use of hydrocortisone eye drops is effective. The local use of hydrocortisone intranasally is only mildly effective.

2. Status Asthmaticus.

In this serious condition, intravenous hydrocortisone or intravenous corticotropin may be a life saving procedure.

3. Penicillin and Other Drug Reactions, Hives and Angioneurotic Edema.

These maladies are usually self limited and respond well to the corticosteroids.

4. Acute Edema of the Larynx.

Whether the edema is from infection or trauma, the use of the corticoids may prevent the necessity of doing a tracheotomy.

5. Critically Ill and Toxic Patients.

The corticosteroids can tide the patient over while antibiotics and other supporting measures are carried out.

6. Idiopathic Midline Granuloma of the Facial Tissues.

ACTH and the corticosteroids are the most effective treatment in this serious condition.

7. Erythema Multiforme.

In many such cases, lesions in the mouth are the first sign of this disease; hence, the otolaryngologist may be the first to see the patient or may be called in consultation. Corticosteroids with appropriate antibiotic therapy give relief and shorten the course of the disease.

8. Pemphigus.

Like erythema multiforme, the first signs of pemphigus are often lesions in the mouth. Until the era of corticosteroids and corticotropin, this disease has been considered to have a fatal outcome. The author has been consulted in three cases of pemphigus. All were treated with corticotropin, corticosteroids and antibiotics. Two are now well on no therapy and one is free of symptoms on a maintenance dose of hydrocortisone.

9. Histamine Cephalalgia.

If histamine and other vasodilating types of therapy fail to give relief, corticosteroids, and particularly intravenous corticotropin, will often bring about improvement; however, vasodilating therapy should be continued.

10. Contact Ulcer of the Larynx.

Cauterization or fulguration of the lesion followed by a short course of corticosteroid therapy is well justified in this condition. Granulation tissue is diminished, and epithelium can more readily cover the ulcer.

11. Laryngeal Polyps and Extensive Nasal Polyps.

The following regime is recommended in these conditions:

a. Surgical removal of the polyps.

b. A short course of post-operative corticosteroid therapy.

c. Elimination of etiological factors.

d. Appropriate allergic treatment.

12. Fresh Stricture of the Esophagus.

Less scarring results if the corticosteroid therapy is initiated immediately. Standard treatment should also be followed.

13. Eczema and Dermatitis of the External Ear.

Local use of hydrocortisone with the appropriate antibiotic is very effective in these cases.

14. Bell's Palsy.

In early cases of Bell's Palsy, we have had very good results with the use of vasodilators combined with corticosteroids and a low-salt diet.

15. Meniere's Disease.

We are now treating Meniere's Disease in a manner similar to that of Bell's Palsy. That is, a combination of vasodilatation, corticosteroids and low-salt diet. The author's experience is not large enough to comment accurately upon the effectiveness of this form of treatment in Meniere's Disease.

16. Pre-operatively and Post-operatively in Patients With Depressed Adrenal Cortices. (This has been commented upon.)

As a matter of fact, in many operative cases, it is probably worthwhile to give a few doses of corticosteroids before surgery and for a day following surgical stress.

17. Local Spray and Mist Hydrocortisone Therapy in Respiratory Allergy.

We have had only moderate success with this local type of therapy.

18. Post-surgical Edema.

In surgical cases where post-operative edema is often a problem, corticosteroids can be effectively used. The author has found these hormones especially useful following nasal reconstructive surgery.

19. Status Lymphaticus.

This condition is supposedly due to a hypo-activity of the adrenal cortices and may result in death from very mild stress. Death from anesthesia furnishes the most typical example. If intravenous hydrocortisone could be administered early enough, recovery could be expected.

20. Anaphylactic and Other Forms of Shock.

Intravenous hydrocortisone is one of the most effective measures.

21. Emergency Surgery in Debilitated Patients.

When it becomes necessary to do surgery in debilitated patients, stress hormones should be administered just before and a few days following surgery.

22. In Patients With a Poor Stress Mechanism.

In a paper recently given at the meeting of the American Academy of Ophthalmology and Otolaryngology in Chicago, I stated that it seemed to me that there were many patients with a poor stress mechanism, who gave normal findings on all of the tests now available for this function. In other words, there were no present tests sensitive enough to determine this mild hypofunction. I mentioned that these patients could not well tolerate stress of any kind. For example, they would develop a stuffy nose and sore throat when exposed to drafts, fog, emotional stimuli and so forth. They were subject to headaches and other vasomotor symptoms. Since that time, one of our colleagues called my attention to an article by Tintera[17] on "The Hypoadrenal State and Its Management". Tintera had observed 200 patients in the above category, and he stated that the regular tests, such as the eosinophil test and checking on the corticoid metabolites in the urine, were always negative. He found that the sugar tolerance test was often abnormal. After eating rapidly absorbed carbohydrates, their blood sugar level would rise to hyperglycemic levels, but this would be followed by prolonged hypoglycemic levels. It was during the latter state their symptoms were more marked. He believed that stresses stimulated their overactive parasympathetic system to stimulate the pancreas to secrete excessive insulin. This kept them in a hypoglycemic state. He recommended the following treatment:

1. Where possible, elimination of stresses,
2. Bellergal to block the parasympathetic system,
3. Whole adrenal extract, and
4. Antihypoglycemic diet.

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WASHINGTON UNIVERSITY, SAINT LOUIS, DEPARTMENT OF OTOLARYNGOLOGY.

Offers a Basic Science Course in Otolaryngology to start Monday, September 17, 1956. Complete information about the course may be obtained by writing to Theo. E. Walsh, M.D., Head of the Department of Otolaryngology, 640 S. Kingshighway, St. Louis 10, Missouri.

ELONGATED STYLOID PROCESS EVALUATION OF SYMPTOMS AND TREATMENT.[*][†]

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Long Beach, Calif.

Case reports of symptomatic elongation of the styloid process have long appeared in the literature, receiving consideration as a medical rarity. In more recent years investigators have attributed a widened spectrum of symptoms to styloid elongation and find that with increased attention the diagnosis is not infrequently made in patients previously labeled as psychoneurotic. This paper is presented in the cause of wider recognition of symptomatic styloid elongation, and to add possibly new symptoms to the syndrome.

HISTORY.

Although ossified stylohyoid ligaments had been reported by anatomists for centuries, Weinlecher, of Vienna, is credited with the first surgical removal of a symptomatically elongated styloid in 1872. Stirling[13] in America in 1896 described three cases, one his wife, later successfully operated; and sporadic case reports continued to appear. In 1932 Thigpen[14] reported 11 cases, with some treated by surgical resection and some by fracturing the elongated styloid outwards by digital pressure through the throat. Clinical results with styloid fracturing were not favorable. Morrison[11] in 1940 stated that styloid fracturing "may or may not influence the neuralgia," and Lederer[9] in 1942 noted that "fracture of the styloid process during operations on the tonsils usually produces annoying symptoms" Reports on styloid resection, however, were good, and Turner[15] in 1936 stated empirically that styloid removal would be followed by cure.

In 1937 Eagle[4] reported excellent results from styloid resection in two cases, but Shambaugh in editorial comment on his

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paper, in the Eye, Ear, Nose and Throat Year Book, sounded a word of caution that neck infections had followed previous cases with reported spread of infection to the middle ear and meninges. Since the antibiotic era, however, no mention of complications has been noted in the literature reviewed.

Since 1940 many new symptoms have been attributed to elongated styloids. Fritz[7] in 1940, and Eagle[5,6] in 1948 and 1949 reported over 250 cases of the Duke University group, many of which had intraoral resections with excellent results. Eagle noted about 12 cases that, as well as local throat symptoms, demonstrated pain of external or internal carotid artery distribution. At surgery actual impingement of these vessels could be demonstrated in some cases, with relief of the symptoms on styloid resection. Eagle attributed this to actual arterial compression, or to pressure on sympathetic nerves in its wall. In 1951 Schmidt[12] added a case of throat tightness, hoarseness, restricted voice range and break, and vocal fatigue in a professional singer cured by resection of bilaterally elongated styloids. Douglas[2] in 1952 reported a case of severe third division trigeminal pain that increased on swallowing, and was relieved by resection of an elongated styloid.

Most writers have done intraoral resections mostly with Crowe-Davis gag exposure under general anesthesia, although Johnson[8] used a combination of sodium pentothal and local anesthetic. Loeser and Cardwell[10] reported one case in 1942 operated by an external incision similar to Mosher's, behind and below the angle of the jaw, where the elongated styloid was more laterally placed producing carotid symptoms, and could not be palpated easily in the tonsillar area.

ANATOMY.

Detailed anatomical studies of styloid ossification and anomalies by Dwight[3] in 1907 described the development of the styloid in the second branchial bar from the cartilage of Reichert, with a proximal ossification center, the tympanohyal, and a distal stylohyal center. The proximal center, according to Cunningham,[1] appears shortly before birth and fuses with the petro-mastoid during the first year. The stylohyal center appears shortly after birth and ossifies slowly, fusing with the upper portion after puberty, or fusion may never occur. In some cases it may become continuous with the inferior cornu of the hyoid bone. Anatomists give normal styloid length as 2.5 cm., and state that abnormally elongated styloids are present in about 4 per cent of persons, occurring bilaterally in about half of these. Only a small portion of these elongated styloids are thought to be clinically symptomatic, however.

A styloid of normal length passes between the external and internal carotid arteries, so that abnormal internal or external deviation may cause pressure on either vessel, even though the process is not elongated. The tractions applied to the styloid by the attachments of the styloglossus, stylopharyngeus, and stylohyoideus muscles and the stylohyoid and stylomandibular ligaments probably play an important role in directing the deviation in growth of the elongated styloid tip. The frequently occurring ram's-horn configuration shown in an anatomical specimen in Fig. 1, correlates with X-ray studies, in the writer's cases, that the proximal anterior deviation may result from traction of the stylomandibular ligament and styloglossus muscle, and the distal downward angulation may result from the pull of the stylohyoid ligament and muscle.

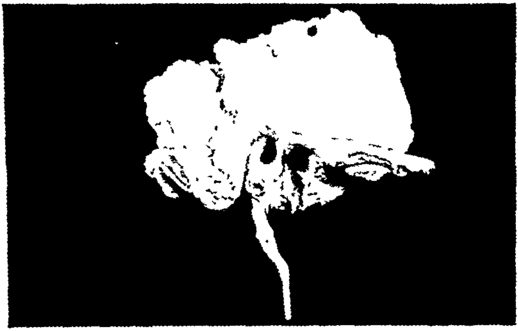

Fig 1 Specimen showing configuration of elongated styloid (From collection of Washington University, St. Louis)

The anatomical specimen in Fig. 2 demonstrates the relationship of the facial nerve and stylomastoid foramen to the styloid process. It was vividly impressed on me during postgraduate training never to succumb to the natural impulse to twist and break off an elongated styloid tip with a hemostat, *if one is found presenting into the tonsil fossa at surgery.* Cases were cited where such a maneuver resulted in backward and lateral fracture of the styloid near the base similar to the breaking of a wishbone, and complete severance of the facial nerve. Although I cannot find an actual report of such an incident in the literature, the mechanics of such an accident, especially with the oblique line of fusion at the base of the styloid shown in Figs. 1 and 2 substantiate this hazard. The writer has taken care to remove styloid tips with minimal *twisting or pressure to the proximal portion.*

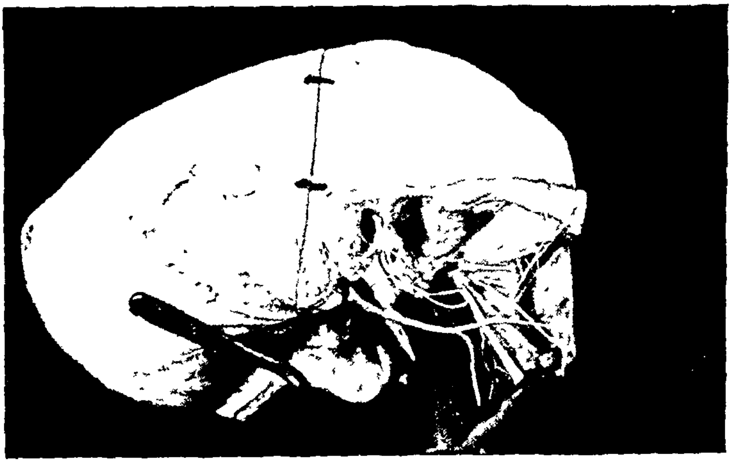

Fig. 2. Specimen showing relationship of facial nerve to the styloid process. (From collection of Washington University, St Louis)

SYMPTOMS.

The most typical history of elongated styloid pain is sticking or soreness in the throat following tonsillectomy with the reaction by the patient that the throat had not healed, though many cases occur long after tonsillectomy, or in unoperated throats. The pain is usually steady, dull-aching and nagging, frequently with increased shooting pain in the throat or to the ear on swallowing. At times a sensation of a foreign body sticking in the throat is noted, or there may be a sensation of drawing or soreness of the throat muscles. Symptoms may follow areas of distribution of disturbed fibers of the Vth, VIIth, IXth or Xth cranial nerves.

The pain attributed by Eagle to internal carotid pressure localizes especially in the parietal and orbital areas, and external carotid pain localizes in the temporal area and the lower side of the head and below the eye.

DIAGNOSIS.

The diagnosis is suggested by the history and is usually made by digital palpation of the firm bony elongated styloid tip in the tonsillar area. As a styloid of normal length does not reach this area, any palpable styloid is abnormally elongated although not necessarily symptomatic. Many elongated styloids are diagnosed on routine examination, or by X-ray, in patients with no symptoms, and their removal is certainly not indicated. In general it has been noted that anteriorly and medially deviated styloids are more likely to be symptomatic, and that more laterally placed elongations, especially those connecting with the hyoid or with a calcified stylohyoid ligament, tend to be asymptomatic. In several of the writer's cases styloids were palpable bilaterally, but were only unilaterally symptomatic on the side of the slightly longer and medially displaced tip.

Consistently in my cases the patient has shown a marked reaction to digital pressure on a symptomatic styloid tip, with such remarks as "that is it" or "that's the spot," and relate that the maneuver caused an exacerbation of pain identical to that of his complaints. X-rays are routinely taken before surgery but were found necessary for diagnosis in only one case where a large tonsil tag was present and the elongated styloid more laterally placed and causing carotid symptoms. In this patient lateral pressure through the tonsil tissue reproduced the symptoms, and though there was a palpable sensation of deep firm resistance the tip could not be clearly outlined.

Granger position anterior-posterior views give the best comparative view of their lengths and their medial or lateral deviation. A lateral view centered behind the angle of the mandible at "soft-tissue" exposure, with the patient's mouth closed and jaw thrust forward so as to flex the neck but extend the head at the atlanto-occipital area gives the best anatomical detail and evaluation of the actual length. Standard lateral X-rays taken for cervical spine, sinus, or skull series may fail to demonstrate large styloids. Fig. 3 shows such a film read as negative for styloid elongation by an excellent radiologist, but a repeated film, taken in the above-mentioned position (see Fig. 4), clearly shows a markedly elongated styloid.

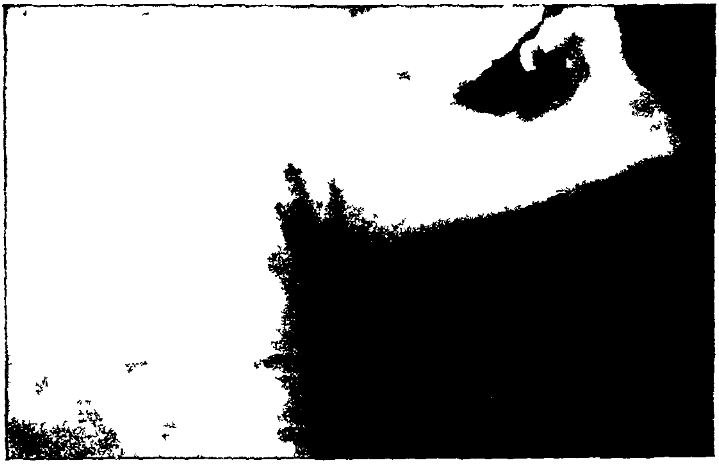

Fig 3 Conventional lateral view of cervical area

Differential diagnostic problems most frequently encountered are: Costen syndrome with mandibular joint disturbances, cervical spine arthritis with radicular pain, atypical migraine, impacted third molars or other dental disease, tonsillar or pharyngeal abscesses or neoplasms, sphenopalatine ganglion neuralgia, and glossopharyngeal, trigeminal or vagal neuralgias.

REPORT OF CASES.

has done seven trans-oral styloid re-
One patient had bilaterally elongated
on the right side, which a right

styloid resection relieved in 1951. Fourteen months later she developed almost identical symptoms on the left side and returned for successful resection of her left styloid. One patient who had mandibular joint tenderness had increasing Costen syndrome symptoms worse with chewing, in the two years since her surgery, though the local styloid tenderness and soreness in the throat were relieved. Four of the patients had had complete tonsillectomies, and two had large tonsil tags. During this same period at least this number of additional patients were seen with palpable elongated styloids who were not operated upon because of absent, mild, or not definitely diagnostic symptoms.

Fig 4 Special lateral view for demonstrating styloid

A 32-year-old woman recently seen in consultation with her neurosurgeon, Charles Spicer, M.D., presented symptoms so interesting as to warrant mention at this time, even though no definite conclusion can be drawn unless planned styloid resection clears her symptoms. Her now-pertinent history is that in November, 1955, she had increasing shooting pains about the left side of her head, followed by sensations of "hot water running over the left side of her head" and a slight sensation of numbness in this area. At this time she had slight drooping of the left eyelid of the pseudoptosis type, and constriction of the left pupil. No definite enophthalmos or unilateral sweating was noted, and complete neurological examination was otherwise noncontributory. X-rays showed markedly elongated styloids of the pseudojointed type bilaterally which were laterally placed. The lateral neck pain was reproduced by deep pressure through her unoperated tonsils, and on deep palpation in a tender area behind the angle of the mandible.

Clinically, this strongly suggests pericarotid sympathetic plexus interference by an elongated styloid as the basis for her Horner's syndrome and pain of carotid artery distribution. This would emphasize sympathetic plexus involvement more than arterial compression in the production of her symptoms. She is the only case I have seen in which external surgical approach may be more advisable than transoral.

SURGICAL TECHNIQUE.

All patients so far have been operated by transoral approach with general endotrachial anesthesia and exposure with the Crowe-Davis mouth-gag and position. In the two patients with tonsil tags the styloids could be exposed easily through a vertical incision behind the tonsil tags, and tonsil tag removal that was not otherwise indicated was not done. Vertical incision was made through the mucosa over the presenting tip, and the styloid exposed by careful blunt dissection with a hemostat and finger palpation. The muscular and tendonous attachments were found most easily and safely separated with a fairly dull flat hook elevator made by bending back the tip of a Freer submucous elevator. When the tip is freed as high as possible, working under intermittent exposure and finger palpation, the distal end is firmly clamped with a tonsil hemostat, to prevent losing it in the pharyngomaxillary space, and the tip resected as high as possible with minimal twisting or side pressure with a long gooseneck mastoid, or universal handle sphenoid rongeur. Usually a 1.5 to 3.5 cm. portion is removed. No bleeding has been encountered, and the incision is closed with about two small plain catgut sutures.

Patients are given wide-spectrum antibiotic coverage for about four days after surgery. One patient with hay fever and sneezing spells developed barely palpable emphysema crepitus in the lateral neck following surgery, but no definite neck infections have developed As a precaution patients are warned not to blow their noses for several days after surgery, to reduce the chance of cervical emphysema or neck infection The operative site heals with little edema of the lateral throat, and the patients experience less discomfort than from a tonsillectomy

SUMMARY

A clinical evaluation of elongated styloid process symptoms and treatment has been presented, with consideration of historical background, anatomy, diagnosis and treatment The possibility that Horner's syndrome may result from styloid pressure on cervical sympathetic fibers of the carotid plexus is suggested

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Professional Building

CONGENITAL HEMANGIOMA OF THE LARYNX.*†

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and

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Hemangiomas of the skin of the face and neck are common tumors in the infant, but are not so common in the larynx. As recently as 1944, Ferguson[1] made a review of the literature and found but seven cases. To these he added one of his own. Dargeon and Daly[2] in 1951, reviewed the literature and found ten cases of hemangioma of the larynx. They reported a case, including the autopsy findings. Since 1951, five infants were seen and treated for congenital hemangioma of the larynx at Babies Hospital. All of these patients had lesions elsewhere on the skin of the face or neck, in addition to the laryngeal hemangioma. They were admitted to the otolaryngological service because of obstructive laryngeal dyspnea. The pathology, symptomatology, diagnosis, and treatment of these cases will be presented.

PATHOLOGY.

Boyd[3] classifies hemangiomas into two main types: capillary hemangioma and cavernous hemangioma. Capillary hemangioma includes port wine stains, birthmarks and strawberry marks. Cavernous hemangioma includes the group made up of structures similar to erectile tissue. They contain large and irregular spaces and lack the definiteness of fully formed blood vessels.

Hertzler[4] has classified hemangioma into three groups: capillary, venous and cavernous. The capillary hemangiomas are intradermal capillary varicosities which may invade the deeper structures. The venous hemangiomas are elevated tumors

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which are present at birth or appear soon after. They may develop rapidly during the first few months of life. The cavernous hemangiomas show large spaces that lack the form of mature blood vessels.[5]

In our group all of the patients had hemangiomas about the face, neck or oral cavity, in addition to a laryngeal lesion. The youngest patient was one month and the oldest four months of age. There were four females and one male, which is different from the cases reported in the past. The literature indicates that males outnumber the females two to one.

Sweetser[6] stated that infantile hemangiomas of the larynx were usually subglottic in location, and in adults the lesions were more apt to be on the vocal cords or above this level. In our series, supraglottic lesions were found as frequently as those in the subglottic area or trachea.

McMahon[7] reviewed Sturge-Weber disease. He reported a case with laryngeal disease. In this disease there are extensive hemangiomatous nevi of the skin and cerebral hemangioma.

There was one pathological report in our group of five patients, and this was a capillary hemangioma. The patient died, and tissue removed at autopsy from the trachea was reported as follows:

> "On cross section, the tissue is dark red in color and homogenous It is hemangiomatous tissue This forms a layer between epithelium and cartilage and is symmetrical The maximum thickness is 3 mm where the diameter of the trachea is 7 mm and one has the impression that when completely filled with blood it probably occluded the lumen completely The histologic appearance of the lesion in the trachea resembles more nearly the so called hemangioendothelioma of the infantile variety, where the lesion appears more solidly cellular with no or relatively few dilated vascular channels"

Hemangiomas vary in size at different times depending upon the blood content of the tumor. This was evident in the repeated direct laryngoscopies on these patients. At times the hemangioma was large, and on other examinations it was scarcely visible.

SYMPTOMATOLOGY.

Suehs and Herbut[8] listed the laryngeal symptoms in patients that had previously been reported. Obstructive laryngeal dyspnea was the outstanding finding. In our group, although the patients had lesions on the lip, tongue and oral cavity, the symptoms of stridor and obstruction of laryngeal origin dominated the clinical picture.

Laryngeal symptoms were not present at birth. As the hemangiomas of the face, lip, tongue or oral cavity became more prominent and appeared to grow, the laryngeal symptoms became more evident. There was noisy breathing, obstructive dyspnea and disturbances in swallowing. The degree of obstruction was variable. At times it was very slight, only to become severe at another period. This was probably due to the changing blood volume of the hemangioma. The patients failed to gain weight. It was necessary to do a tracheotomy in all five patients.

DIAGNOSIS.

The patients were referred to the otolaryngological service because of laryngeal symptoms. The presence of hemangioma in the larynx was determined by direct laryngoscopy. In this group of five infants, two were subglottic and tracheal in location, and the remaining three involved the epiglottis, aryepiglottic folds and the mucosa over the arytenoid cartilages.

The appearance of the hemangioma by direct laryngoscopy varied, depending upon the blood content of the tumor. In each instance the lesions were large enough to produce laryngeal obstruction. There were no biopsies taken, due to the danger of hemorrhage. A pathologic report was obtained on the patient who died. The lesion was classified as a capillary hemangioendothelioma.

TREATMENT.

Hendrick[9] discusses the therapy of vascular tumors. He includes solid carbon dioxide snow, irradiation, electrosurgery, injection of sclerosing solution and tattooing or neutralization of color. The method of therapy depends upon the age of the patient, type and location of the lesion and its rate of growth.

Kasabach and Donlan[10] reported two cases treated successfully by Roentgen therapy.

TABLE I.

Case	Location of Tumor	Hemangioma Elsewhere	Method of Diagnosis	Treatment	End Result
E W	Supraglottic	Face and Lip	Laryngoscopy	Tracheotomy Radiotherapy	Decannulated
L B	Subglottic, Tracheal	Mediastinum	X ray Bronchoscopy	Tracheotomy Radiotherapy	Good but mediastinal hemangioma still enlarging
C C	Supraglottic, Base of tongue, Epiglottis and aryepiglottic folds, trachea	Skin and structures of jaw, neck and pharynx	Laryngoscopy	Tracheotomy Radiotherapy	Died suddenly Hemangio-endothelioma
J S	Supraglottic Subglottic	Neck, ear and soft palate	Laryngoscopy Bronchoscopy	Tracheotomy Radiotherapy	Decannulated
A.V.	Supraglottic	Lip, tongue, pharynx, Eustachian tubes, middle ear, aryepiglottic folds and arytenoids	Laryngoscopy	Tracheotomy Radium Radiotherapy	Good

TABLE II.

Case	Sex	Age in Months	Outstanding Symptoms	Duration
E.W.	F	4	dyspnea	7 days
L.B.	M	2½	dyspnea	8 days
C.C.	F	1	dyspnea	1 day
J.S.	F	1	dyspnea	2 days
A.V.	F	1¼	dyspnea	2 weeks

X-ray therapy was selected for the laryngeal lesions in our group. This type of treatment is not without danger to the infant larynx. It is important to keep the dose low and to spread it out over a period of several months. Each patient received an average of 400 Roentgens within a period of nine days. This was repeated at intervals of three months until a total of 1200 units had been given. There was no evidence of any X-ray complication to the larynx as a result of this therapy. The lesion appeared to become smaller after completion of the treatment.

The response to X-ray treatment is difficult to evaluate. Capillary hemangiomas have a tendency to disappear spontaneously after a few years. It is possible that patients might do just as well if the laryngeal lesions are left alone. An exception would be the lesion that is growing rapidly. One of our group has involvement of the mediastinum as well as the larynx and trachea. It has not been possible to control the mediastinal lesion with X-ray therapy.

All of the patients needed a tracheotomy. These patients required a long stay in the hospital and a great deal of nursing care. In order to cut down the expense involved, the parents were taught how to change the tracheotomy tube and to take care of the patient at home.

RESULTS.

Four of the five patients are living. One died of respiratory obstruction, and at autopsy the lesion was found to involve a large portion of the trachea. Two patients have been decannulated. One will be decannulated in the near future.

The remaining case has extensive involvement of the mediastinum in addition to the larynx and trachea. The prognosis in this patient is questionable.

The lesions that are present on the face will require further therapy which will be supervised by the plastic surgeon.

SUMMARY.

Congenital hemangioma of the larynx is probably more common than is indicated by reports in the literature.

Tracheotomy is necessary if laryngeal obstruction is present.

X-ray therapy may be of value in these lesions. Precautions are necessary when it is used.

The prognosis is good if the lesion is not too extensive and if a vital structure is not involved.

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A RARE CASE OF CHRONIC OTORRHEA WITH INTRACRANIAL COMPLICATIONS.*

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C. M., a 73-year-old man, came to the office because of a running ear. Within three months he had developed intracranial complications that caused his death. The diagnosis was not made until the autopsy findings were completed. Later it was learned that there are laboratory tests whereby an accurate diagnosis might have been made earlier.

A brief review of this patient's clinical course will bring out some of the diagnostic problems encountered:

The patient was a retired teamster who had had an intermittent discharging right ear since childhood. Another otologist reported that the ear was dry one year before, but that it had been discharging six weeks before the patient first consulted me on October 6. As he had a large amount of granulation tissue in the middle ear, some of this tissue was removed for microscopic examination. This tissue was reported as being a granuloma. X-rays of the mastoid made at this time showed them to be sclerotic, with no definite bone destruction. He was treated at the office for the next three weeks, but as he had an increasing amount of pain and headache, he was hospitalized on October 28.

His internist had treated him for some time for diabetes, which was controlled with insulin. He also had a mild hypertension. Medical and neurological examinations were otherwise essentially negative. A lumbar puncture and spinal fluid were normal. X-rays of the mastoid on October 28 showed no change from those made on October 6. He was given 8,000,000 units of Penicillin within the next six days.

A radical mastoid operation was done on October 30. The

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soft tissues over the mastoid and ear were very vascular and somewhat indurated. The mastoid cavity was extremely small, and there was very little pus present. Granulation tissue in the mastoid antrum and middle ear was removed for microscopic examination. This tissue again revealed a granuloma. The dura was exposed and found to be normal. There was no evidence of extension over the lateral sinus area. There was no VIIth nerve paralysis immediately following surgery.

His postoperative course was very stormy. He had chills and fever, and his headache persisted. X-rays of the chest revealed a pneumonia, which responded to treatment within a week. He began to develop weakness of the VIIth nerve and had difficulty in swallowing; he also had some hoarseness. There was a paralysis of the right side of the palate as well as the right vocal cord. A lumbar puncture and spinal fluid were again negative. At times he was fairly alert mentally, but at other time he was stuporous.

On November 7 a neurosurgical consultation reported that there was no evidence of increased intracranial pressure, in spite of the fact that the VIIth to the XIIth nerves on the right side were involved. The neurosurgeon stated that such lesions are most often due to malignancies extending along the base of the skull and originating in the nasopharynx or immediate vicinity. He found no evidence of a posterior fossa abscess or mass. He noted that there was slight tender swelling at the tip of the mastoid, and he suggested that later a biopsy might be taken from a gland in this area.

On November 9 the blood culture was reported negative. The sensitivity tests of cultures made from the ear showed the organisms to be most sensitive to Chloromycetin. As previously stated, the patient had received 8,000,000 units of penicillin during the first six days of hospitalization. The penicillin was stopped and 250 mg. of aureomycin was given every four hours for one week. When the ear culture showed the organisms to be sensitive to chloromycetin, he was given 250 mg. of this drug every six hours for the next ten days; and Gantrisin for two weeks; however, none of these drugs produced a definite improvement in the patient.

His general condition gradually became worse perature was irregular, although not very high. He had so much difficulty swallowing that it was necessary to feed him through a Levine tube. He had a constant headache. At times he was lethargic or stuporous. X-rays of the skull on November 18 were reported as showing no evidence of involvement of the petrous tip and no evidence of destruction at the base of the skull. These X-rays did suggest a mass in the nasopharynx. (See Fig. 1.)

Because of the patient's poor mental and physical condition it was difficult to examine the nasopharynx well, and a definite tumor mass had not been seen. The pathologist who performed the autopsy six weeks later reported that no mass could be palpated in the nasopharynx. On November 20, with strong retraction of the soft palate, a small definite mass about one cm. in diameter was seen high in the vault on the right side of the nasopharynx. The surface of the epithelium seemed intact. A large amount of this rather soft friable tissue was removed for microscopic examination. This tissue was reported as a granuloma, similar to the three specimens previously removed from the ear canal, the mastoid antrum and the middle ear. Several pathologists examined these specimens, and all agreed on the diagnosis. On December 18 X-rays of the skull were repeated; the report stated that no change had occurred in the past month, and that there was no evidence of involvement of the petrous tip or foramina at the base of the skull. (See Fig. 2.)

The patient's condition rapidly became worse, and he expired on January 6, three months following my first examination. Fortunately, an autopsy was done. The following is a brief resume of the autopsy findings relating to this case:

"A mass about 3 cm. in diameter and 1 cm. thick, with very ill-defined borders, surrounded the entire right petrous tip area and extended into the foramen magnum. This mass enveloped the cranial nerves of the right side. It had dissected downward near the mid-line to a point just above the nasopharynx on the right but apparently had not penetrated through the lining of the nasopharynx on this side. As previously noted, palpation of the nasopharynx by the autopsy surgeon revealed no abnormality. The bony petrous area was mushy in consistency and had the same type of material as

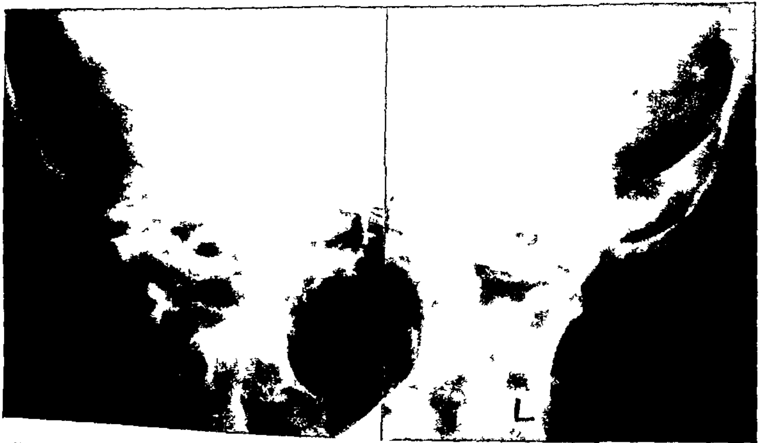

Fig 1 X-ray of the petrous ridge on December 18th 3 weeks before patient's death showed no evidence of bone destruction although the autopsy showed almost complete destruction of this area

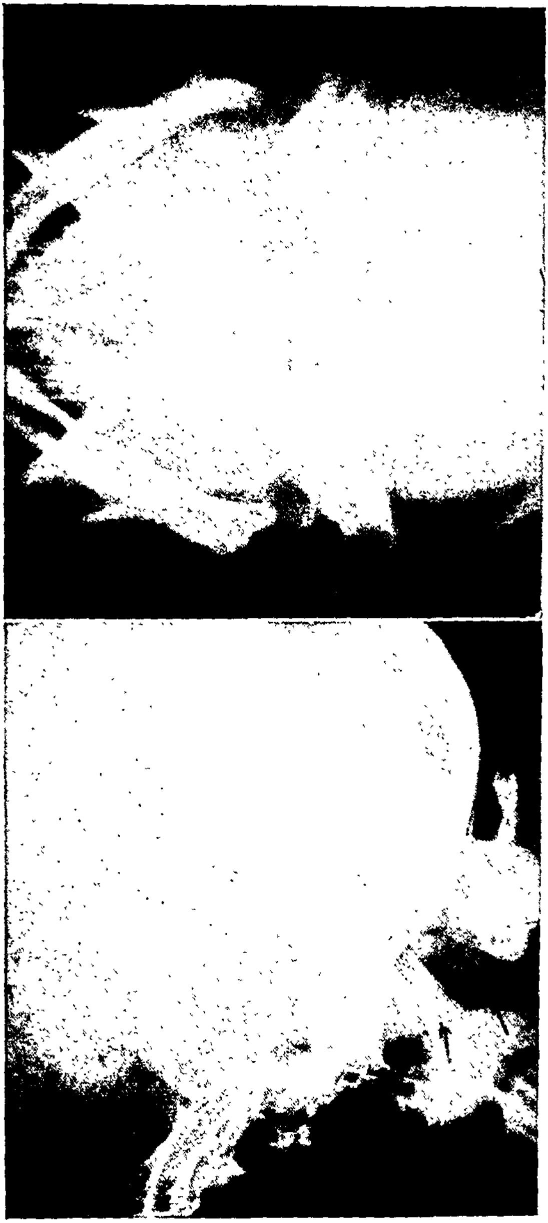

Fig. 2. X-rays of the skull on December 18th, 3 weeks before patient's death, showed no definite evidence of involvement of the foramina of the base of the skull. A mass in the nasopharynx was "suggested" by the Roentgenologist, but this is not seen clearly in the X-rays.

the mass, which was a thick, rubbery, reddish-yellow mass, resembling granulation tissue. There was also a thrombus of the right lateral venous sinus. The gross impression was that of a neoplasm, but when the microscopic sections of this mass were examined, they revealed many areas with very typical actinomycosis." (See Fig. 3.)

Actinomycosis of the ear is very rare. Risch[1] in 1938 reviewed the literature and found only 31 cases involving the ear, and only 12 of these cases involved the middle ear. Nowack[2] reported two cases in 1938. Townrow[3] reported a case in 1945 that was remarkably similar in all aspects to my case. Schubert[4] reported one case in 1951, and Bistrenin[5] reported a case.

The fact that actinomycosis of the ear is so rare is of academic interest, but it is of much more interest to our specialty that actinomycosis, as well as other diseases that produce granulomatous lesions, are rather commonly found in the mouth, nose, throat and neck. The fact that a differential diagnosis can be made between actinomycosis and these other diseases, by making bacteriological studies of this granulomatous tissue, makes it imperative today that we are familiar with these tests in order to determine which specific drug or antibiotic is to be used.

Months later I learned that laboratory tests might have enabled us to make this differential diagnosis earlier. While attending an instruction course given by Thomas B. Fitzpatrick, M.D., Professor of Dermatology at the University of Oregon Medical School, we were told that whenever one had a chronic granuloma which had not been identified, this tissue should be ground up and cultured for a fungus, etc. He was referring to an article written by Lyle A. Weed, M.D., and David C. Dahlin, M.D.,[6] of the Bacteriology and Surgical Pathology Division of the Mayo Clinic.

Dr. Weed and Dr. Dahlin reported 14 cases illustrating some of the common errors made in trying to establish an etiological diagnosis of tissue removed when only a histological examination was made. Their work was based on the generally known facts that certain organisms elicit a fairly characteris-

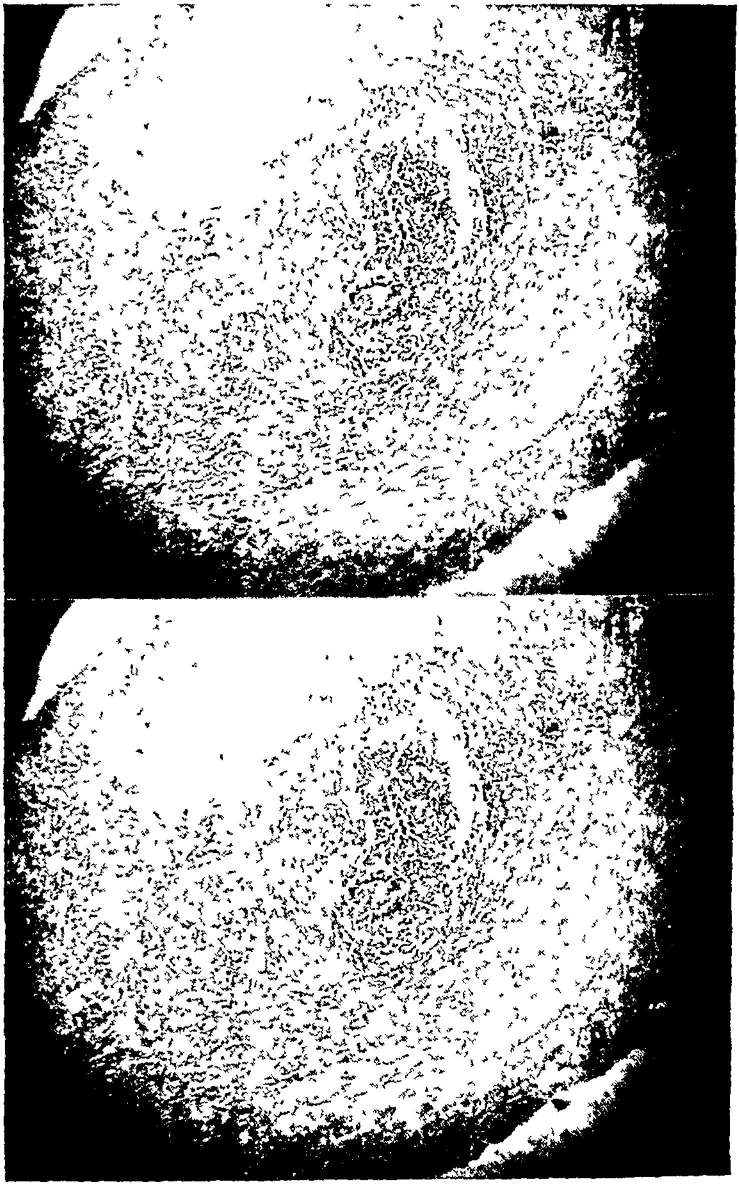

Fig 3. Microscopic examination revealed many areas of typical Actinomycoses with the characteristic sulfur granule arrangement

tic histological response, although experience has also proven that different organisms may stimulate reactions that are histologically indistinguishable and, on the other hand, that a given species of micro-organisms can produce a wide variety of histological patterns. These authors for some time had made extensive bacteriological examinations of tissues removed surgically, as well as the histological examinations of the tissues removed. These studies revealed many discrepancies that occur between the histological interpretation and the results of the bacteriological studies of the same tissue.

Briefly, their method of making these bacteriological studies is to have as much of the tissue as possible removed by the surgeon under sterile technique, if possible. They preferred to have abscesses aspirated rather than to use swabs. The tissue should be ground to a paste in a sterile mortar and diluted with sterile saline or broth to make a 10 to 20 per cent emulsion. It is not satisfactory to use pieces of tissue for culture or inoculation. This emulsion is then cultured on the various media that may be required, which any modern bacteriological laboratory will be able to do.

The following is a brief summary of the conclusions of these authors:

"Tuberculosis was frequently not recognized from the biopsy alone, but bacteriological studies of the same tissue gave an accurate diagnosis. It frequently may be impossible to establish a diagnosis of blastomycosis or coccidioidomycosis by histological methods alone because of the absence of organisms, or because the organisms present may not possess adequate identifying characteristics. Brucellosis may be recognized in the tissue only by isolating the organism, as it gives no characteristic histological pattern. Actinomycosis is probably frequently confused with certain fungus infections, but careful attention to the size of the filaments would many times avoid this error; however, actinomycosis can be distinguished with certainty from nocardia only on culture. Because of the inherent limitations of histological procedures, we believe that every specimen removed for biopsy should be studied as thoroughly as possible by acceptable bacteriological methods in an attempt to establish the etiological agent. Such procedures, if performed diligently and carefully, should result in more accurate diagnoses and thereby prevent needless operations and save many lives by indicating the proper therapy."

COMMENT.

It was very disconcerting to treat a patient for three months without making a diagnosis. Had we made bacteriological studies of the tissue taken for biopsy we might have made the diagnosis before death, and, as penicillin is effective in the treatment of actinomycosis, we might have saved his life.

CONCLUSIONS.

1. A case report of a patient who had actinomycosis of the ear with intracranial complications that caused his death in three months, has been presented.

2. The diagnosis was not made until the autopsy findings were complete.

3. Biopsies taken from the middle ear, mastoid antrum and nasopharynx revealed a granuloma.

4. Bacteriological studies made from granulomatous tissue are necessary to make a differential diagnosis between the various diseases that produce such granulomatous tissue.

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1010 B Street.

THE ADENOID AND ITS RELATION TO THE EAR.*†

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It is significant that the term adenoid merely means resembling a gland, while the term adenoids, by the simple addition of the letter "s", implies a pathologic state.

Adenoid disease has been recognized since the time of Hippocrates. Meyer of Copenhagen, when he published the classic description of adenoid vegetations in 1868, called attention to the "adenoid facies" exhibited in portraits and busts several centuries old. It is curious, therefore, that this disease and its management should be so generally neglected, both in the teaching of otolaryngology and in textbooks on the subject. The neglect, however, helps to explain why the removal of the adenoids is looked upon as one of the simplest and easiest of all surgical procedures, which it is not; in fact, the generally casual attitude toward the operation helps to explain, in its turn, why the results which follow it are so often less than satisfactory.

The adenoid was once thought to be a completely functionless structure. There is now an increasing belief that the growing child requires nasopharyngeal lymphoid tissue for present and future protection against infection. When, however, adenoid tissue has become hypertrophied, it not only cannot fulfill its normal function, but it also serves as a focus of infection.

PATHOLOGY.

The most important subject in diseases of the pharynx is the pathologic changes in the lymphoid tissue. There are two principal types of pathological changes to which the adenoids (lymphoid tissue) are subject: *a*. Hyperplasia; *b*. Hypertrophy with infection.

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In hyperplasia (see Fig. 1), which usually occurs in infants and young children, there is a marked reproduction of the normal lymphoid cells with very little, if any, of the growth of

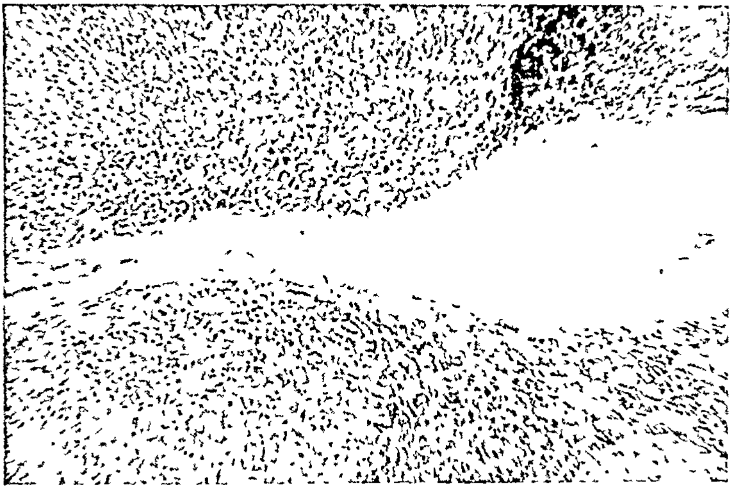

Fig 1. Demonstrates simple hyperplasia showing prominent enlarged follicles

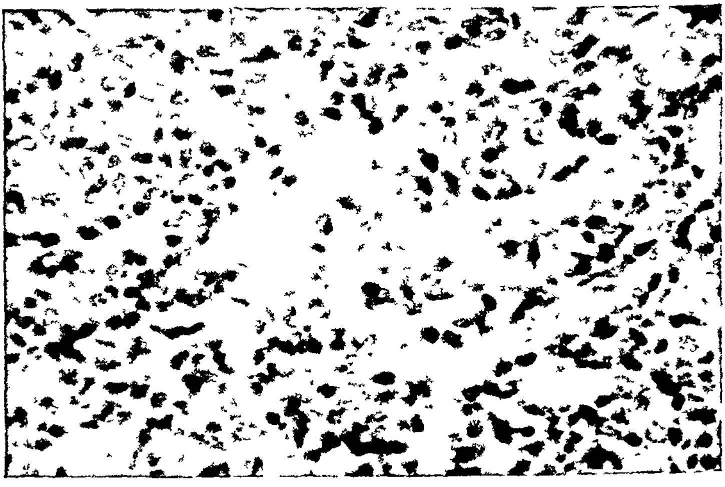

Fig 2. Shows hypertrophy with infection This shows chronic inflammation with many small congested blood vessels and increasing amount of fibrous tissue Pycnosis is present (this means degeneration of the cells that stain black) Exudate and degenerated material in cells of the adenoid are also present in this illustration.

connective tissue (see Fig. 2). This tissue is subject to infection, and then the second type of disease occurs, in which a marked increase in vascularization takes place, with the other elements of inflammation, such as round cell infiltration and fibrous tissue formation. The folds within the lymphoid masses

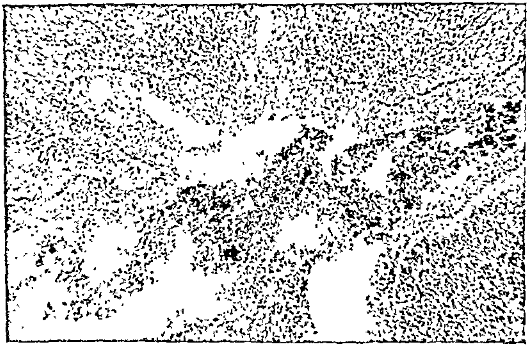

Fig. 3. Shows a crypt of the adenoid with distention of the crypt and an exudate present; upper respiratory epithelium was also present.

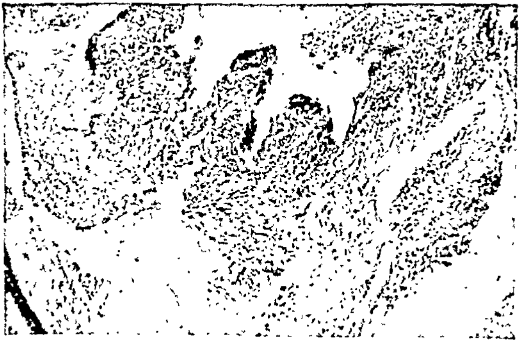

Fig. 4. Chronic inflammation, fibrosis and pycnosis are present; few lymphoid cells; fibrous tissue degenerating; cysts spaces.

in the adenoid harbor the infected material. The bacteria in the crypts find their way into the follicles. As the process of recurrent acute inflammation in the crypts and lymphoid tissue occurs, so does the retention of the infectious material within the crypts increase (see Fig. 3). This retention causes these crypts to dilate with accumulated masses of degenerated epithelium in the crypts, clumps of bacterial flora, particles of food and mucous (see Fig. 4). Slowly this hypertrophic process produces atrophy by squeezing out the lymphoid tissue. The toxins from the crypts (distended) or bacteria are absorbed into the circulation by the blood stream, or lymphatics. A marked anemia develops from diseased adenoids.

AUDITORY IMPAIRMENT.

The presence of diseased adenoids may be followed by a number of serious consequences, some infrequent but some very frequent indeed. They include snuffling, nasal obstruction, mucopurulent or purulent nasal or postnasal catarrh, absence of nasal resonance, laryngeal stridor, suppuration of the accessory nasal sinuses, cervical adenitis, gastrointestinal disturbances, defective growth and backwardness, aprosexia or inability to fix the attention, neuroses, systemic infections, and a number of other unfortunate results.

In particular, neglect of diseased adenoids may have an extremely serious effect upon the structure and functions of the ear. Deafness which follows obstruction to the Eustachian tube by adenoid tissue may occur by several mechanisms: the membranum tympani may be indrawn or retracted; postnasal catarrh may spread along the Eustachian canal, with resulting acute and chronic otorrhea. From the aural point of view, the most dangerous variety of diseased adenoid tissue is the soft, diffuse type, which usually is not excessively hypertrophied. This type of tissue more than any other type, is apt to become inflamed and suppurative, and to cause middle-ear disease and eventual auditory impairment.

These are not imaginary or even occasional possibilities. They are serious considerations which must be constantly borne in mind. It has been said with good reason, that a definite loss of hearing can practically and without difficulty always be demonstrated in a child with hypertrophied adenoids who is not properly treated before the age of 10 years. One British observer reported diseased adenoids in 62 per cent of 375 school boys ranging in age from 6½ to 12½ years, 27 per cent of whom had some impairment of hearing. Another observer found auditory damage in 70 per cent of all children with diseased adenoids, while still another found that 60 per cent of all hearing impairment in children could be attributed to this cause.

These hearing difficulties arise for the most part during the period of active growth. Even if atrophy of the hypertrophied adenoid tissue should take place by natural processes, the regression cannot be counted on to occur before the child's hearing is in danger of being irreversibly damaged. It should also be remembered in this connection that the atrophy of the adenoid tissue which so often seems to occur at the age of puberty is usually only apparent; the adenoid tissue itself does not shrink, it merely seems smaller because, except in occasional cases, the nasopharynx has increased in size.

The otolaryngologist, confronted with the question of whether or not to operate for adenoids, often has a difficult decision to make. There are two circumstances, however, in which his course of action is clear: *1.* If enlarged adenoids are present in association with chronic suppurative otitis media, they should be removed without delay, even at the risk of an acute flare-up of the aural disease. *2.* If auditory impairment is present in association with even minimum adenoid vegetations, an operation to remove the adenoid tissue must be undertaken without delay.

EXAMINATION OF THE NASOPHARYNX.

Examination of a child suspected of harboring hypertrophied adenoids is not always easy; in fact, examination with the mirror is practically impossible in children under six years of age, and if the symptoms and clinical signs are not conclusive, examination with the finger may be necessary. If the finger is passed up behind the soft palate, a soft mass, which feels almost like a bundle of worms, may be found filling, or almost filling, the nasopharynx. When the adenoids are diseased, the finger, when it is withdrawn, will be covered with tenacious mucus streaked with blood. If no blood is present, it can be assumed that the nasopharynx is normal. If digital examination can possibly be avoided it is well to omit it in young children, particularly nervous children, to whom it is always very disturbing.

Posterior rhinoscopy should be employed routinely in older children. If adenoids are present, the amount of enlargement can be determined by the extent to which the upper portion of the posterior edge of the septum is hidden from view in the mirror. In extreme hypertrophy the entire septum is invisible. Nasopharyngoscopy should also be performed routinely in all older children.

A child with adenoids is likely to present such findings as a high-arched palate; irregular eruption of the second teeth, which project forward and overlap; and blobs of mucus or a mucopurulent discharge dripping from the postnasal space.

TECHNIQUE OF REMOVAL OF THE ADENOIDS.

Unless the removal of the adenoids is carried out with careful attention to technical details, portions of the hypertrophied structure will inevitably be left *in situ*, particularly in the following locations:

1. The angle formed by the roof and posterior wall of the nasopharynx. One frequently finds, when examining the patient after operation, that while the main mass has been removed, a fringe of tissue has been left behind.

2. Posteriorly, behind the soft palate. When the soft palate is raised, a tag of tissue attached to the mucosa is likely to be found. Sometimes it is large enough to be seen without lifting the palate.

3. Laterally, in Rosenmüller's fossae, above and below the Eustachian cushions. When the adenoids are soft and diffuse, tissue is particularly likely to be left behind in this location.

The operation is performed under general anesthesia, which is continued in the deep plane, with abolition of the pharyngeal reflex, until the whole procedure has been concluded. The patient is in the recumbent position, with the head down and slightly flexed. A mouth gag is used, as in tonsillectomy.

Opinions differ as to the most useful instruments for removal of the adenoids. There is rather general agreement that a curette is preferable to the various kinds of postnasal forceps formerly employed, because it removes the growth more completely, as well as more quickly. An essential step in the curette technique is the use of a horizontal incision, made with a knife, through the mucosa immediately below the adenoid. This precaution prevents stripping of the nasopharyngeal mucosa as the curette is removed. Otherwise, surgeons tend to develop their own preferences. Some prefer the Beckmann-Thomson curette, others the LaForce adenotome, which works on the guillotine principle and which is fitted with a cage which receives the excised tissue and prevents its lodging in the larynx or being swallowed. Still others prefer the LaForce and Kelly adenotomes used in combination.

The following technique is simple and satisfactory:

1. Before any operative procedure the nasopharynx is palpated digitally, to determine accurately the size, character and position of the adenoids. In particular, it is noted whether the fossae of Rosenmüller are clear, whether the Eustachian cushions are normal or enlarged, and whether the atlas is unusually prominent.

2. The central mass is attacked first, with the LaForce adenotome. Usually this step removes only the upper two-thirds.

3. The Kelly direct vision adenotome is next inserted and the lower part of the adenoid mass is cleanly removed. Any lateral portions are also removed under direct visualization.

4. Finally, the Love retractor is inserted, and the fossae of Rosenmüller are inspected. The Yankauer speculum may also be used for this purpose. Any lymphoid tissue remaining in these areas is removed with a punch forceps. It is sometimes useful to use a Eustachian catheter at this stage of the operation.

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The operation is performed under general anest[illegible] is continued in the deep plane, with abolition of which, because of its size or location or both, is producing symptoms, the preferable method of management is secondary surgery. The symptoms that we encounter from adenoid remnants include: *1*. conductive deafness; *2*. recurrent colds; *3*. tinnitis (a subjective symptom) ; *4*. pressure and ear sensation (a subjective symptom) ; and *5*. aerotitis.

It is almost impossible to examine the adenoid region in young children with a nasal pharyngoscope or laryngeal mirror; therefore, to be positive in your diagnosis, it is wise to give the patient an anesthetic and examine the nasal pharynx thoroughly. Where tissue is found we remove enough to ventilate the Eustachian tube and the nares. Often hidden infection is found in the pharyngeal recess as small cysts. Head colds frequently start in this area. Remember to remove the tissue around the torris by nibbling with the punch forceps.

On the surface, irradiation seems a satisfactory method of managing recurrent hypertrophy of the nasopharyngeal lymphoid tissue, since lymphoid tissue ranks next to the sex cells in its sensitivity to this physical agent. On the other hand, this method is not without danger, and reported results vary widely. It seems wisest, therefore, to reserve it for small lymphoid accumulations in and around the Eustachian tubes in patients whose chief complaint is conductive deafness. Before it is resorted to, it is essential that the diagnosis of conductive deafness be established; more than one patient with nerve deafness has been submitted to unnecessary irradiation because the proper diagnostic methods were not first employed.

X-RAY TREATMENT.

The X-ray treatment is of value in real young children in whom you are sure no large mass of adenoid tissue is present. The technique for X-ray treatment of the Eustachian tubes or lymphoid tissue in the nasal pharynx is as follows:

The output of the machine (with these factors) : 48 Roentgens (r) per minute. The treatment is through two fields, a right and left lateral, each 7x7 cm. in size located to crossfire the nasal pharynx. The treatment is one weekly for four weeks with both right and left fields treated at weekly intervals. The treatment to each field each week is two minutes

5. The last step is a systematic inspection of the whole nasopharyngeal area, including first the roof, then Rosenmüller's fossae and the Eustachian cushions, and finally, the posterior wall. If the operation has been correctly performed and all adenoid tissue has been removed, the entire surface will be perfectly smooth.

Bleeding is sometimes so brisk during removal of the adenoids as to hamper the procedure. It seldom fails to stop, however, within a minute or two of the conclusion of the operation, and under normal circumstances, the total loss is not more than one or two ounces. If bleeding does not cease spontaneously, a thorough examination must be made at once, under good illumination, to see whether a tag of lymphoid tissue has been left behind. It is astonishing how often profuse bleeding occurs from this simple cause, and how promptly it ceases when the tag has been removed. If bleeding still continues after the removal of the tag, the quickest and most effective way to control it is to insert a gauze plug into the postnasal space. In some cases it is necessary to suture.

POSTOPERATIVE BLEEDING.

In cases of postoperative bleeding, a postnasal pack is inserted in the usual manner, making sure that you have a string from the pack coming out of the mouth and attached to the cheek. This string coming from the mouth is essential for easy removal the following day. A pack should never be left in place over 24 hours. If bleeding recurs after the pack is removed, it is much better to put in another pack than to leave the original pack in place. A pack in place over 24 hours invites secondary infection. Hemostatic drugs are also useful in controlling minor postoperative bleeding.

RECURRENCE.

When operation is performed carefully, by the technique described or some similarly precise technique, recurrence of adenoid tissue does not take place in more than 10 per cent of all cases. Some recurrences are undoubtedly instances of physiologic hypertrophy of remnants of adenoid tissue rather than true regrowth. No treatment is necessary in this type of recurrence. If, however, one is dealing with a true regrowth,

SUMMARY.

Removal of the adenoids should be advised only when there is no doubt that the hypertrophied lymphoid tissue is producing symptoms, since it is now known that the growing child requires this type of tissue for present and future protection against infection If, however, it can be established that impairment of health is related to the presence of adenoids, operation should be performed promptly It should also be performed without delay if auditory impairment is associated with even minimum adenoid vegetations A precise technique is essential The most important cause of recurrence is inadequate surgery at the original operation. If postoperative recurrence of nasopharyngeal lymphoid tissue gives rise to symptoms, secondary surgery is usually preferable to irradition

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SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

Washington, D C, May 5-10, 1957.

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RADIUM TREATMENT.

Radium treatment is used in older children, and the monel metal applicator is used. It is important to apply the radium applicator so that the tip of the applicator will fit directly over and in contact with the orifice of the Eustachian tube. The dose is 50 mg. of radium for 12 minutes each side (36,000 mg. per second). Seventy-five per cent of the Beta rays are absorbed in the first three millimeters of tissue. Never use the radium applicator for longer than 12 minutes, and never give more than four treatments in a period of six weeks, and no repeats. You must treat before irreversible changes occur in the middle ear. With the radium we destroy the trigger zone and lymphoid tissue that acts as a focus of infection; this contributes toward raising the patient's resistance, and the patient has fewer colds. It is important to remember the following: *1.* Use radium only in selected cases; *2.* Place radium in its proper place; *3.* Treat early before irreversible changes occur; *4.* The duration of symptoms is important in the prognosis; *5.* To get the best results, use in conductive deafness, tinnitis, pressure in the ear, aerotitis, and repeated cold cases.

Improvement in hearing follows proper attention to the nasal pharynx only in conductive deafness; however, improvement in hearing can occur in the absence of any form of treatment (spontaneous).

RESULTS.

There are few surgical procedures in which results are as good as they are after surgery of the adenoids providing *1.* that the operation is indicated and, *2.* that it is properly performed. The dull listless and even stupid-looking child becomes bright, active and healthy-appearing, with a new enjoyment of life. The hearing impairment caused by obstruction of the Eustachian tubes by adenoid tissue also promptly disappears since, when operation is performed promptly, this type of deafness is reversible.

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THE
LARYNGOSCOPE.

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SYMPOSIUM.

The Operation for the Mobilization of the Stapes in Otosclerotic Deafness.

PARTICIPANTS.

PHILIP E. MELTZER, M.D., Boston, Mass.
JOHN R. LINDSAY, M.D., Chicago, Ill.
VICTOR GOODHILL, M.D., Los Angeles, Calif.
GEO. E. SHAMBAUGH, JR., M.D., Chicago, Ill.
CLAIR M. KOS, M.D., Iowa City, Ia.
EDMUND P. FOWLER, JR., M.D., New York, N. Y.

MODERATOR.

HOWARD P. HOUSE, Los Angeles, Calif.

REMARKS.

SAMUEL ROSEN, M.D., New York, N. Y.

OPEN DISCUSSION.

BARRY ANSON, M.D., Chicago, Ill.
FRANZ ALTMANN, M.D., New York, N. Y.
RICHARD BELLUCCI, M.D., New York, N. Y.
JOHN J. SHEA, JR., M.D., Memphis, Tenn.
SHIRLEY BARON, M.D., San Francisco, Calif.
L. F. MORRISON, M.D., San Francisco, Calif.
J. H. THOMAS RAMBO, M.D., New York, N. Y.

DR. PHILIP E. MELTZER: The members of the panel agree to speak extemporaneously. I do not have the facility of speaking in that manner; therefore I wrote these notes last night. Forgive me if it sounds a little didactic, but this is the only way that I can present what I have to say in a suitable manner.

I am grateful for the privilege of appearing in this symposium. I am comparatively inexperienced, though well acquainted with the history of this procedure under discussion. In this country it had its inception at the institution where I had my otological training; in fact, it was under the very man who in 1892 introduced stapes mobilization, namely, Dr. Frederic L. Jack.

His chief, Dr. Clarence Blake, also experienced in this procedure and a most erudite individual, summarized the several surgical efforts of that era to improve hearing. In this review he related the attempts by Kessel, Schwartze, Urbantschitsch, Boucheron, Miot and Gelle.

Deafness then as now occupied the minds of these great otologists. It was the deafness resulting from suppurative otitis that concerned them mostly. Chronic otitis insidiosa, as otosclerosis then was known, was vaguely understood. Politzer was perhaps one of the first to describe stapes fixation clinically as a result of a bony ankylosis. Haberman demonstrated histologically what this malady really was.

The surgical efforts in vogue from 1875 to 1888 to improve hearing may appear absurd to you in retrospect, but at that time they were considered bold and daring. The procedures began with simple incision of the membrana tympani, advancing courageously to the removal of drum membrane, malleus and incus. Then began the attack on the stapes itself, first severance of adhesions, then severance of the stapedial tendon, finally the revolutionary procedure of stapes mobilization, including stapedectomy.

Boucheron was perhaps the first actually to practice mobilization, but to Miot must go the real credit of enlightening the otologists of that period about this operation. With the exception of modern incisions to expose the tympanum he described with infinite detail the entire procedure of stapes mobilization, the indications, pitfalls and results.

To these early French and American pioneers our salute! After the initial reports of success with this operation came the years of silence. Nothing more was seen or heard about mobilization in our lifetime. Why? I can only venture a guess. It failed; hearing was not lastingly improved.

This generation is grateful for the perseverence of Holmgren, the surgical ingenuity of Sourdille and Lempert, and now for the astute observation of Rosen, and once again to Lempert. Why Lempert again? Ask yourself, would the tympanum be exposed so beautifully without his incision for tympanoplexy?

To Rosen must go the credit of rediscovery of stapes mobilization. Rosen's recent technique has definitely given otologists a new enthusiasm.

You have observed that I have refrained from quoting figures for immediate, temporary or late results. I have too vivid a recollection of my humble early results with the fenestration operation to do so, and I am too old a hand to sound off prematurely; furthermore, what difference does it make what these early results were? Does it matter if three or four patients are improved by stapes mobilization in ten attempts? Are not three or four successes a real attainment? Given time and experience and understanding as to why we fail, should we not expect better results just as we improved our results with the fenestration operation? I believe so.

I am too affectionately involved with the fenestration operation to ever abandon for a moment my first love. I would be negligent in my duty if I were not progressive enough to adopt a procedure, the success of which my own eyes have witnessed.

The hearing improvement brought about by stapes mobilization is of such magnitude that I confidently look forward to its becoming as firmly established a procedure for the improvement of hearing in otosclerosis as the fenestration operation.

We acknowledge Dr. Rosen's contribution regardless of whether my prophecy proves to be correct.

DR. HOUSE: Thank you very much. I think it is very apropos to review the historical developments that led up to our modern clinical surgery in the treatment of otosclerosis. I will ask Dr. John Lindsay to discuss with you some of the histopathological activities which occur in and about the footplate and which influence the stapes mobilization procedure.

DR. JOHN R. LINDSAY: Dr. Anson has demonstrated the development of the normal stapes in the human. In my part of this symposium I wish to illustrate mostly by slides some of the problems that are presented by otosclerosis as it causes fixation of the footplate. There are two ways in which one may observe the effect of otosclerosis at the joint: one is by direct observation at the time of operation; the other and more effective way is to study the microscopic sections cut through the stapes and the joint. In our collection of otosclerotic material there are not many cases of stapes fixation; therefore, it seemed best if I mentioned the literature freely to illustrate to you some of the different ways in which the joint may be attacked. I have selected illustrations by Guild, Eggston and Wolff, Lempert's collection, Nager and Meyer, Anson and Bast, some of our own, and perhaps one or two others.

The first slide is from an illustration from Dr. Guild which illustrates certain points. Three sections are shown from a case with fixation of the stapes where there was a hearing loss of about 55 db in speech frequencies. It illustrates several factors: one is the approximate comparison of the thickness of the crus with the footplate of the stapes in the part where it is affected by the disease. If you look at a section like this, you may not get a reliable idea of how firmly fixed that stapes may be; you see the ankylosed part in only one dimension. In this case Dr. Guild stated that the fixation occupied the posterior part of the joint instead of the anterior, which is usually involved, and the entire posterior third of the circumference of the joint between the footplate and the labyrinthine capsule was involved; hence this would probably be a difficult case to mobilize, if you were dependent upon the stress which could be applied through the crura from the head or neck.

Dr. Rosen illustrated a group of 50 normal stapes, in one or two of his articles, which gives an idea of the variations in the normal. The next illustration is one of Lempert's collection showing a diseased stapes. Presumably this shows otosclerotic involvement of one crus of the stapes and gives some idea of how the strength of one crus or perhaps both crura might be reduced by the disease.

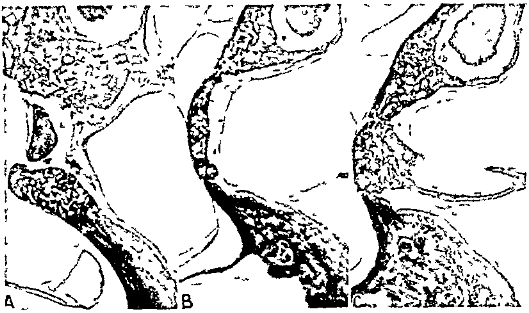

Fig 1 From Guild, Stacy R, Ph D: Histologic Otosclerosis Annals of Otology, Rhinology and Laryngology, 53 246-266, 1944 A, B and C are, respectively, photomicrographs of vertical sections through the anterior, middle and posterior thirds of the oval window of the left ear This otosclerotic area was actively growing and had formed osseous connections with the stapedial footplate in three regions, two of which are shown See Test Aut No 10692

It is a clinical observation that in some cases of early fixation or with a moderate degree of deafness from otosclerosis there may be slight mobility of the stapes when you first touch it with an instrument. The question is whether the fixation is all bony or is it partly fibrous. This illustration is from Dr. House's case which was reported a few years ago. It was successfully fenestrated, but on microscopic examination when the case came to termination a few years later we were unable to find any definite bony fixation. This suggested that the degree of fibrosis at the joint may be partly responsible for the *impairment of hearing and for the fact there may be slight* movement in some of these cases. There is a possibility, of course, that during fenestration the removal of the incus may have loosened the stapes.

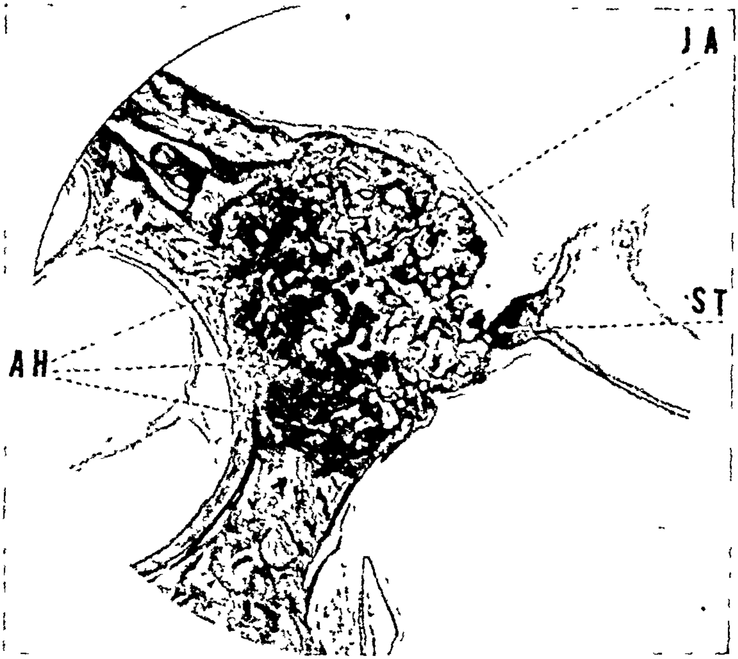

Fig. 2. From Nager, F. R., and Meyer, Max: Beiträge zur normalen und pathologischen Histologie der knochernen Labyrinthkapsel. Passow-Schaefer, Beitrage Bd 30 (1932) (Abb. 65. Fall 46. 3½ year old boy. Page 235). Magnification ×20. H. & E. stain. Active part of the focus (JA) at the margin of the oval window niche with extension to the stapes (ST) Older part of the focus (AH) in the form of compact "Breccie" at some distance from the niche. Sharp demarcation from the primary labyrinthine bone.

This next illustration shows a thin, well sclerosed bridge of bone producing the ankylosis. When viewed in this plane alone it would look like an ideal case for mobilization; perhaps it is, but one section does not give a complete picture. From this section alone one does not know how extensive the ankylosis may be around the circumference of the joint. This picture also gives only a partial idea of the relative strength of the crus as compared to the strength of the footplate. The footplate is a broad structure, relatively thin but broad, and may be stronger than it would appear on this view; also the crus may be broader in one dimension and, therefore, stronger than it appears here.

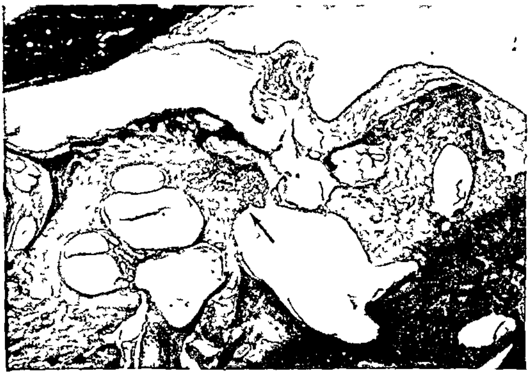

Fig 3 From House Howard P, Perret, Pierre and Lindsay, John R Histologic Observations on Fenestrated Human Ears Part II Otosclerosis. Two Years Following Fenestration Supplement to the Transactions of the American Academy of Ophthalmology and Otolaryngology, July, 1951 Given before the Otosclerosis Study Group, October 7, 1950 Horizontal section through the cochlea and stapes footplate between which is seen a large focus of otosclerotic bone (arrow) Stapes ankylosis is not present

(Slide) These next two illustrations again showing fixation are taken from Bast and Anson's book. I call your attention to the point with which everybody has been concerned, how long a stapes is going to remain mobile after it has been mobilized. Many people, including myself, have been surprised with the length of time that they may remain mobile. Perhaps it is in the case that, with a well sclerosed bridge of bone, when it is broken loose there may be a longer period of freedom from reankylosis. On the other hand, as you see in some later slides, if an active focus extended across the joint and produced the fixation one might expect to get an earlier reankylosis. There may be practical significance to a very strong Schwartze's sign, as our best clinical indication of activity around the joint. Such a case might not be so good for mobilization as one in which there was no activity, and the ankylosis was of sclerotic bone.

(Slide) In this next case the ankylosis is formed by a thick bridge of bone. It illustrates the comparative thinness of one crus and how easily a crus might be broken in trying to break loose such a bridge of bone.

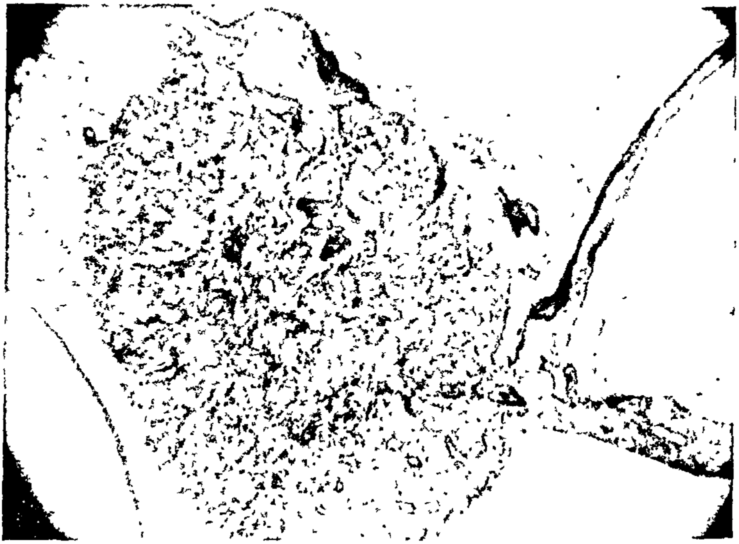

Fig. 4. From Eggston, Andrew A. and Wolff, Dorothy: Histopathology of Ear, Nose and Throat. Williams and Wilkins Co., Baltimore, 1947, p. 456, Fig. 278. Otosclerosis as seen in H and E stain showing the differentiation between normal and pathological bone. Note ankylosis of the footplate of stapes and the flecks of calcium deposited in the scar tissue lying between the crus and the cochlear wall.

(Slide) The illustrations to follow represent rather extensive cases of otosclerosis, advanced cases. It is, therefore, doubtful whether cases like these might come up for mobilization since a severe hearing loss might be expected. In this case you can see an active focus spreading across the footplate, and you see also the thin crus. You can understand the difficulty in mobilizing such a footplate if you were depending upon the force that you could apply by way of the head or neck transmitted through the crura. In more advanced cases it may be necessary to have some means by which the footplate can be mobilized directly, if we hope to have success.

(Slide) I call your attention to the visibility of the footplate of the stapes. In a horizontal section through the temporal bone, through the stapes, one gets the impression that the stapes footplate would be easily visualized. Another point illustrated here is a dehiscence of the bony canal for the facial nerve in proximity to the posterior crus. It would be quite easy to injure the nerve in the middle ear portion where it

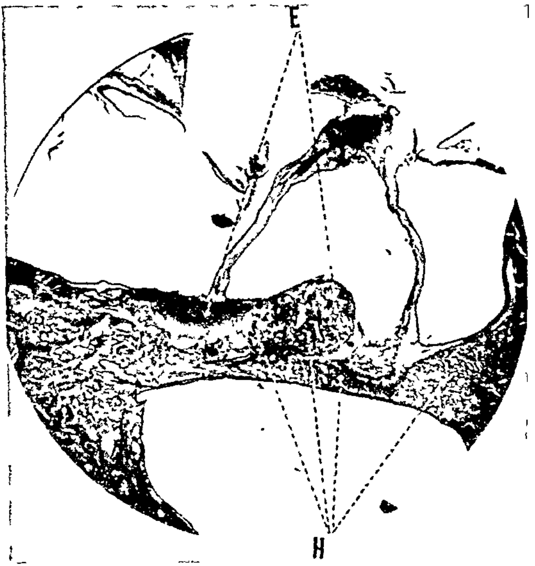

Fig 5 From Nager F R, and Meyer Max Beiträge zur normalen und pathologischen Histologie der knöchernen Labyrinthkapsel Passow-Schaefer Beitrage Bd 30 1932 (Abb 63 Fall 27 p 231) Magnification ×13 H & E stain Submucosal exostosis (E) extending over the oval window from its anterior border An otosclerotic focus (H) extends from behind over the stapes footplate into the exostosis

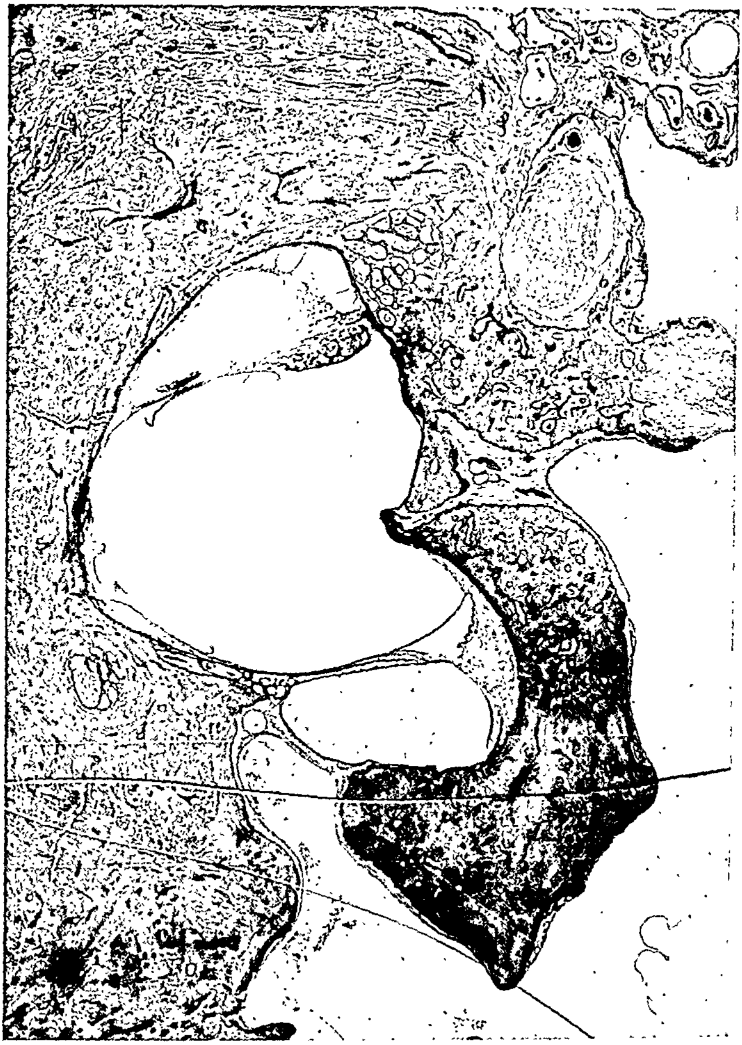

Fig. 6. Female, age 104. Photomicrograph of vertical section through the stapes. Advanced otosclerosis with total loss of hearing for air and bone conducted sound. Marked ankylosis, sclerotic focus of otosclerosis. Round window niche was patent. Profound deafness due to inner ear degeneration secondary to otosclerosis.

borders on the niche of the oval window. If you look at the stapes on the vertical section it looks quite different.

This illustration from a vertical section shows the depth of the niche down to the footplate. The view of the footplate at operation is partly obscured by the borders of the niche, also by the fact that directly over it are the crura and the incus. The view which it is possible to get of the footplate is somewhat limited.

This next case of ankylosis is from a patient who was totally deaf at the time of death. The following two illustrations also show extensive otosclerosis. They illustrate how hopeless it might be to expect to get a good mobiliaztion in the really advanced cases. Here you see a large otosclerotic focus extending across the footplate, and to expect to mobilize it would be hopeless. The next three illustrations also show advanced otosclerosis at the footplate and are cases in which any attempt at mobilization would be hopeless.

DR. HOUSE: Thank you, Dr. Lindsay. I am sure you can visualize now some of the problems which we attempt to face when we surgically mobilize the footplate.

I should like now to call on the next participant of our symposium, Dr. Victor Goodhill. Now the remaining members of our symposium will speak more on the problems pertaining to indications, what they themselves tell patients pertaining to mobilization surgery, what they are doing in the way of tricks and techniques, following which we will go into the summary of the results.

DR. VICTOR GOODHILL: Stapes mobilization is neither a standard operation nor is it applied to a standard disease. Stapes mobilization is used in the surgical approach to relieve stapes ankylosis in what we call for convenience, "clinical otosclerosis." This stapes ankylosis may be due to histological otosclerosis, about which we know too little, or to other bone diseases, some known, some still unknown. Dr. Lindsay has pointed this out clearly.

In conductive deafness of presumably otosclerotic etiology, exploratory tympanotomy is now indicated. We may find other lesions, such as fibrotic periostitis, ossicular dislocations, ossicular or fenestral anomalies, etc. If such exploration reveals footplate ankylosis, the stapes mobilization approach is physiologically logical.

The objectives of stapes mobilization in such cases are dual: *1.* Restoration of near normal mobility to foot plate; *2.* Maintenance of integrity of the middle ear mechanism (which contributes approximately 26 db of impedance matching normally).

The indications require new and broader audiologic conceptions. Basically, the surgical approach is indicated if pre-operative bone conduction measurements indicate the possibility of restoration of air conduction thresholds to the 30 db average level. Sometimes it is possible to hope for this level when bone conduction is lower than 30 db, *i.e.*, 35 db or 40 db, or even 45 db. Bone conduction, in my opinion, is usually an indicator of *cochlear function,* which is at least at the same level, and sometimes even better than the bone conduction level; but there is another indication for mobilization, and that is in certain types of profound mixed deafness, presumably of basic otosclerotic etiology where the bone conduction would be too low to hope for serviceable restoration of hearing. In such cases (b. c. levels of 45 to 55 db) it is possible to hope for improvement to a level where hearing aid performance can be greatly enhanced by improved discrimination, diminished feedback, and elimination of recruitment, which is so commonly found in high level deafness.

The patient is told that the procedure may or may not be successful; that it may be successful in one ear and not in the other; in both, or in neither. If, by the usual criteria, the patient is also a candidate for fenestration surgery, he is told that fenestration remains as a secondary possibility in case of failure or persistent reankylosis. Since the audiologic indications of this approach are broader, obviously *not all* of these patients are *going* to be fenestration candidates in the event of failure.

Now some remarks about technique—Based upon the two principles enunciated, footplate motion must be obtained, and normal middle ear function must be maintained.

This is an approach requiring multiple techniques because of multiple-pathologic states One cannot operate on an "all or none" law Application of force must be guided by quantitative techniques, obtained only from surgical audiometry.

In this approach one must be especially aware of the surgical rule of *non nocere* in planning surgical steps This is decidedly not simple surgery I will not go into the details of my technique, which has been described in several publications I would like only to stress some aspects Local anesthesia and the trans-canal approach are used The incision is a generous one for several good reasons Adequate removal of bone from postero-superior quadrant for good exposure is an obvious necessity in many cases The use of the microscope for examination of details is extremely valuable, but in many situations it has been found that the microscope interposes a mechanical obstacle to the delicate manipulations necessary in this surgery There are many delicate maneuvers needed in the various procedures and steps of this approach The microscope unfortunately seems to interpose a solid object which does not allow for complete flexibility or fluidity of motion

The actual surgical approach consists of a progression of steps, depending upon the gross pathological findings

1 If palpation reveals a stable incudostapedial joint and rigid crura, then one or two procedures may be effective in obtaining lysis of the ankylosis

a The trans-incudal digital approach uses a small needle shaped probe anchored within the periosteum of the lenticular process of the incus *Digital vibrato force is then applied* If lysis cannot be obtained, then this step is followed by

b The use of a small electrically controlled micro vibrator placed in the same trans-incudal position

2 If we find on palpation that the incudo stapedial joint is loose, but the crura are still rigid, the application of force is transferred to the capitulum of the stapes The lenticular process is moved very slightly away from its capitular attachment, so that *a.* The needle probe may be inserted into the capitular surface of the stapes and force applied by digital techniques, or

b. The micro-vibrator may be used if digital techniques alone are inadequate.

3. If palpation reveals atrophic or non-rigid crura, then the only approach possible is a direct footplate approach. I believe Dr. Lindsay's slides pointed this out most eloquently. In many of these cases the footplate is a deformed structure, and the crura are delicate, slight, senile bony struts, which are very easily fractured. In these cases it seems desirable to go to the footplate. Techniques used on the footplates today are extremely varied and highly experimental. One may use a probe, an electric or pneumatic hammer, the new intertia vibrator, a chisel, or any other instrument. They are all, at present, in preliminary experimental phases. I would like to point out that simple puncture of the footplate will frequently produce a spectacular improvement in hearing on the operating table, which will be a very encouraging step temporarily; however, in many cases this spectacular improvement *(step 3)* will disappear completely or partially when the drum is closed (*step 4* of surgical audiometry). The improvement in hearing one would expect with closure of the drum does not occur, but there is actually a drop in hearing. In these cases, it is my opinion that one has done nothing but a temporary puncture-fenestration of the footplate, one which in most cases is temporary and destined to immediate closure. Here we see another reason for the necessity of surgical audiometry, in that it gives us details of middle ear physiology otherwise unobtainable.

If the footplate maneuver is successful and is accompanied by physiologic continuity of the mobilized fragment with either one or both crura, one may hope for a significant gain in hearing.

Closure details are important. Meticulous removal of blood is important. Blood clots may ruin an otherwise excellent mobilization. Precise gentle canal packing is important in preventing postoperative oozing and excessive postoperative edema.

Surgical problems merit a few words. Incudostapedial dislocation is certainly not a desirable accident and should be avoided whenever possible. Whenever it occurs the incus should be carefully repositioned upon the capitulum. The same philosophy applies to crural injuries, whether they be complete fractures or green stick fractures. They should be avoided. If they occur, the fragments should be repositioned wherever possible. Bleeding should be controlled by electrocoagulation and packing, because, as I mentioned before, it may be a significant postoperative problem.

Vertigo is usually not a serious problem in this surgery. There is frequent inconsistency between the occurrence of vertigo on the operating table and what happens to the hearing. Usually there may be momentary vertigo at the time of successful mobilization, but it is possible to have no vertigo with marked improvement in hearing. It is possible to have a great deal of vertigo with no improvement in hearing.

Perforations are not infrequent. They usually are small, slight tears. In my practice I always close them with a small thin free skin graft, and they are invariably healed at the end of a week.

Antibiotic therapy is of course advisable to prevent otitis media and labyrinthitis. We use the mycin drugs.

The packing is removed on the sixth or seventh day. Occasionally tubal inflation may be necessary three or four weeks postoperatively.

I feel strongly that surgical audiometry is a necessity for this surgical approach. I do not have time to go into details, but some type of relative measurement is necessary in guidance. For this purpose we have been using the surgical audiometric *nomograph*, which has been recently described as a guide to stapedolysis surgery.

I should like to point out that reankyloses do occur, and the percentage is difficult to estimate at this early stage. Reankyloses may occur at any stage of the postoperative period; they may occur very early, within several weeks, several months, or even a year later. It seems that they are more common following footplate techniques than when the approach is through the incus or through the stapedial capitulum.

Can reankylosis cases be re-operated? Yes, they can, and in some cases they may be more successful in a secondary procedure than in the first procedure.

As far as candidate selection criteria are concerned, I must conclude at the present that any otosclerotic patient with a marked conductive deficit, with reasonably decent bone conduction, is a suitable candidate. Preoperative bone conduction, air conduction, air-bone gap, age, and other factors have been studied. I have been unable to come to any conclusions regarding these factors as significant guides to candidate selection.

In summary, in clinical otosclerosis, as we conceive of it today, stapes mobilization is a useful procedure. It is a precise micro-surgical approach to the stapedial vestibular joint. It should be a tool in the hands of the otologic surgeon who is trained and experienced in all phases of temporal bone surgery including fenestration. It is extremely early; in its present status there are no final well defined standardized techniques.

In all of our modifications and improvements, however, two things must be remembered. We are trying to accomplish:

1. Restoration of motion to the ankylosed footplate, and

2. Maintenance of the integrity of the middle ear mechanism.

DR. HOUSE: Thank you, Dr. Goodhill. I will ask Dr. George Shambaugh to present his portion of this symposium.

DR. SHAMBAUGH (showing of movie): This movie depicts the current technique which Dr. Derlacki and I are using. I emphasize, it is our current technique because this is an operation which is very much in the developmental stage. We are constantly changing our technique, and I am sure as time goes on everyone else will be changing his also.

Three things are emphasized in this technique: one is direct visualization of the footplate; second, the use of the operating microscope, which we think is essential for the best results; and the third is the use of pressure directly inward toward the vestibule as our principal attack on attempting to mobilize the stapes. This technique, which we are showing, is a combination of Dr. Rosen's, Dr. Kos', Dr. Goodhill's and Dr. House's, with a few ideas which Dr. Derlacki and I have added.

We tell our patient with otosclerosis who is ideally suited for fenestration that he has a choice of a new experimental and very uncertain procedure called stapes mobilization, or a very well established, dependable and relatively certain procedure of fenestration. We then give him, as well as we can, the advantages and disadvantages of these two procedures, and we do it in writing so that he may take home the information and at his leisure consider it and make up his mind as to what he would like to have done. The advantages of stapes mobilization, we tell him are, first of all, its simplicity as far as he is concerned, with only three days away from work as compared to three weeks with fenestration in the average case. Usually there is very little vertigo compared to fenestration. The hearing improvement, if successful, is comparable to that obtained by fenestration and occasionally, but only occasionally, exceeds that obtainable by fenestration, and finally, and we think most important, there is no postoperative cavity requiring care the rest of that patient's life.

The disadvantages of stapes mobilization, we tell him, are first of all, the complete unpredictability and uncertainty of the result. We have no way of knowing until we get into the middle ear as to what type ankylosis he has, and whether or not it will be suitable for mobilization. Second, as yet we know nothing of the permanence of the result of this procedure. Third, the chances of failure are at least as great as the chances of success, so that in all probability this patient will have to come back for subsequent surgery. We are very conservative in our promises to these patients. We emphasize that this is a preliminary surgical procedure only, and he should consider it as such. If he is very fortunate it will be successful, and he will not need another operation; but he should be prepared to return for the fenestration operation six months later. This is extremely important, because psychologically these patients tend not to try a second operation if the first operation has failed.

We remove quite a bit of the bony rim in the average case, 3 or 4 mm. in many instances, in order to get an excellent view of the footplate from above. Nearly always, in spite of the narrowness of the oval window niche, we can obtain a view of at least half of the footplate. We sacrifice the chorda tympani very often and explain to the patient ahead of time, "You must expect the sense of taste to be deranged for a few months."

The footplate as seen through the operating microscope has a bluish color; when normal, white and interlaced with vessels when replaced by otosclerotic bone. We use magnification of 16 times for most of our manipulation. We believe audiometric control of the procedure is extremely important. Our first test is made with the tympanic membrane replaced after elevation, when we have a traumatized, relaxed membrane closing the middle ear cavity. This test is our base, to which we compare any improvements obtained by our manipulation.

Our first attempt at mobilization is through the lenticular process of the incus, using a blunt ended mobilizer that we call the "Derlacki mobilizer" rather than a sharp needle. The needle frequently will split the incus or head of the stapes. Our chief direction of force, using intermittent pressure, is directly inward. That is the direction in which the crura are constructed by nature to take the greatest force without fracture, and we have had our greatest success in obtaining mobility by this maneuver. If the lenticular process fractures we then transfer pressure to the head of the stapes, watching for movement of the footplate itself. In some cases we can see a line of fracture in the footplate, often with escape of perilymph.

I would estimate that in about half the cases that we are able to mobilize we do so working through the incus, and in the remainder through the head of the stapes. Rarely do we go directly to the footplate. We are still very hesitant about this, because of the possibility of labyrinthine damage. It should be remembered that the chance of making the hearing worse by mobilization is three times greater than by fenestration.

A good many of the cases that are audiometrically improved on the table will drop down within the next month. After one month it is less common to have the hearing decline, but gradual loss of the improvement due to refixation of the stapes can occur many months after an initially successful result.

DR. HOUSE: Thank you very much, Dr. Shambaugh. I am sure that this emphasizes again the value of visual education when you are attempting to describe techniques; indeed a very beautiful film.

I will now call on Dr. Kos to give his thoughts on stapes mobilization.

DR. C. M. KOS: Few surgical procedures of comparatively recent history have aroused such professional imagination and intense interest as the current employment of stapes mobilization. Judging from the frequency with which this procedure is being used and the numbers of those who are investigating its value, it should not be long before current uncertainty and skepticism may be swept aside or confirmed.

For the present, patients who accept the recommendation of stapes mobilization should be thoroughly apprised of the uncertainty of receiving hearing improvement. This uncertainty cannot be couched in terms which underestimate the incidence of failure. There is nothing about the otoscopic examination or the audiometric findings which permits me the latitude of predicting a given result.

It seems reasonable to inform the patient that there is some chance of recovering practical and serviceable hearing, and that the likelihood of residual damage or injury to the ear structurally or functionally is a very remote one.

While they are rare, the risks and complications seem to be directly proportional to the ability, training and experience of the surgeon. He should be qualified and prepared to give definitive otological treatment, and to accept the responsibility of managing any problem that may arise in connection with his efforts. The most common risk is in increasing the hearing loss due to fracture of the crura, excessive trauma to the stapes, or postoperative adhesions. This may add 10 to 15 db to the preoperative impairment and consequently increase the intensity of the tinnitus.

Other complications may include tympanic perforations, otitis media and labyrinthitis. Appropriate caution, a meticulous technique and protective antibacterial therapy should reduce these to an extremely low incidence.

Facial paralysis is possible, but it can be avoided if carelessness and incompetence can be eliminated. Temporary paresis or paralysis may result from an intent to infiltrate the anesthetic agent too widely and too deeply.

The eardrum may become undesirably flaccid because of an imperfect replacement of the epithelial cuff or because of subepithelial bleeding.

Postoperative bleeding may be annoying but not of a serious consequence.

Injury to the chorda tympani nerve may cause the complaint of perverted taste in a few instances.

Should the mobilization procedure prove ineffective, the patient should know at the outset whether the fenestration operation may be the next step required to achieve hearing improvement.

In the event satisfactory hearing improvement results from stapes mobilization and then regresses, the procedure may be repeated, and so far as is currently known, may be repeated indefinitely at six to 12 months' intervals.

A variety of techniques may be required to bring about release of the ankylosed footplate. Sometimes the pulsating pressure applied to the neck of the stapes, as described by Rosen, will suffice. In many instances a special pick inserted into the head of the stapes through the lenticulo-capitular capsule has given excellent results.

This approach (slide) is personally preferred for the firm leverage it exerts on the footplate through the crura. Upon mobilization the epithelial cuff is replaced, and a thin strip or a compressed strip of gel foam is placed over the incision; I do not use packing. The patient is in the hospital one day, arriving in the hospital the night before the operation is done, and he is discharged at four o'clock that afternoon.

In other instances mobility may be obtained by exerting pressures on the lenticular process, such as Dr. Shambaugh and Dr. Goodhill described. It may be necessary to apply the picks in a prying manner to the edges of the footplate in order to secure adequate mobilization. In others, a mechanical device such as that used by Dr. House may be directed at the footplate so as to fracture or even pulverize the structure.

It is apparent, from these few technical preferences, that the search continues for effective and safe techniques which may increase both the incidence and durability of successful results. It is rather obvious at this point that the demonstration of some success has stimulated an inquisitive drive and inspired a general curiosity which may lead to better solutions; but this is not the stage in the development of this procedure for unlimited enthusiasm.

DR. HOUSE: Thank you very much, Dr. Kos.

Dr. Ned Fowler will now close the participants' portion of this program, except for their responses in discussion.

DR. EDMUND P. FOWLER, JR.: I regret that the other panelists did not go into the indications for mobilization of the stapes more clearly since this was part of Dr. House's charge to us. We have discussed the indications, however, and I think that most of us seem to agree that mobilization procedures are indicated on any case which is suitable for fenestration; they also can be done on patients who are so advanced in years that fenestration is unwise. Mobilization procedures can also be done on patients who do not wish a major surgical procedure, or the inconvenience of a radical mastoid cavity, and also on patients where the bone conduction is down so much that a successful fenestration operation will not bring the hearing to a practical level. Closing the air-bone gap in severe deafness will often just bring the hearing to the 30 db level or will occasionally permit the wearing of a hearing aid with comfort.

Dr. House admonished us to tell exactly what we say to each patient. I tell each patient that two out of three cases get a bad result; that *he* is, therefore, likely to get a bad result. Now the reason I say "two out of three" is because I really don't know how many bad results there are going to be in the future, although I have some theories on the matter which I shall relate later. I try to emphasize that two patients out of three get a bad result, because I wish nobody to sue me or come in with a blackjack, or complain that I have minimized anything; however, I do bring out the facts that very few cases have any complications. There are a few patients that have a slightly increased hearing loss, from the operation, especially if there has been much manipulation of the footplate; and there are a few patients who have lost their hearing completely, or almost completely. The statistics on this phase will be brought out later by Dr. House. Since the worse ear is usually operated, a slightly increased hearing loss will, as a rule, be of comparatively little consequence. I always discuss fenestration and further rehabilitation with a hearing aid with the patient, and point out that there is very little chance that the hearing will be made sufficiently worse by mobilization that a fenestration cannot be done in the future.

If the patient asks me how much discomfort there will be I usually tell him, "about as much as having a wisdom tooth out." Having a wisdom tooth out can be quite a serious procedure, as some of you know, and I think that some patients have much less trouble than others, but I believe that comparison with molar extraction is fair, from the standpoint of the patient. The standpoint of the surgeon is quite another matter, for I do not believe that mobilization procedures are simple; on the contrary, I think they are more difficult than fenestration.

At the Columbia-Presbyterian Medical Center we have made a series of observations and have developed a different technique from those described this morning. Incidentally, I work in such close collaboration with Dr. Franz Altmann and Dr. Milos Basek that I hardly know where their ideas leave off and mine begin in this discussion of our philosophies and the technique that we have developed in our laboratory. After months of observations we feel reasonably certain that mobilization of the stapes with the Rosen techniques and the modifications of them used by Goodhill and House do not break up an ankylosed joint. We believe that mobilization is best attained by footplate fracture and deliberate severance of the anterior crus of the stapes. Our reasons for this position are as follows:

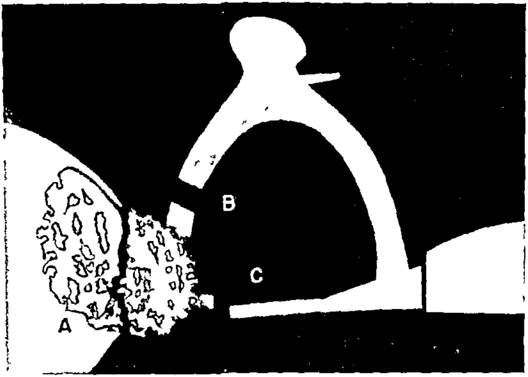

Fig 1 Diagram of otosclerosis focus in anterior border of oval window in horizontal section Mobilization through "A" impossible, or if accomplished would soon be followed by re ankylosis Anterior crurotomy "B" with footplate fracture, 'C' brings about mobilization through the posterior crus

(Fig. 1 typical otosclerosis with ankylosis of the anterior part of the stapes with heavy cancellous new bone). With this type of otosclerosis I cannot see how one can possibly mobilize the stapes through the vestibulo-stapedial joint, in the manner suggested by the writings of most authors on the subject of mobilization.

(Fig. 2.) In this kind of ankylosis where there are calcifications of the annular ligament from otosclerosis, I should think that if you shake the ankylosis loose, either with a vibrator, hammer or pick, that the patient who says "doctor, what is going to keep this thing from rehealing" has a point. This sort of calcified joint is likely to be reankylosed just as any other joint in the body, if loose calcium spicules are present.

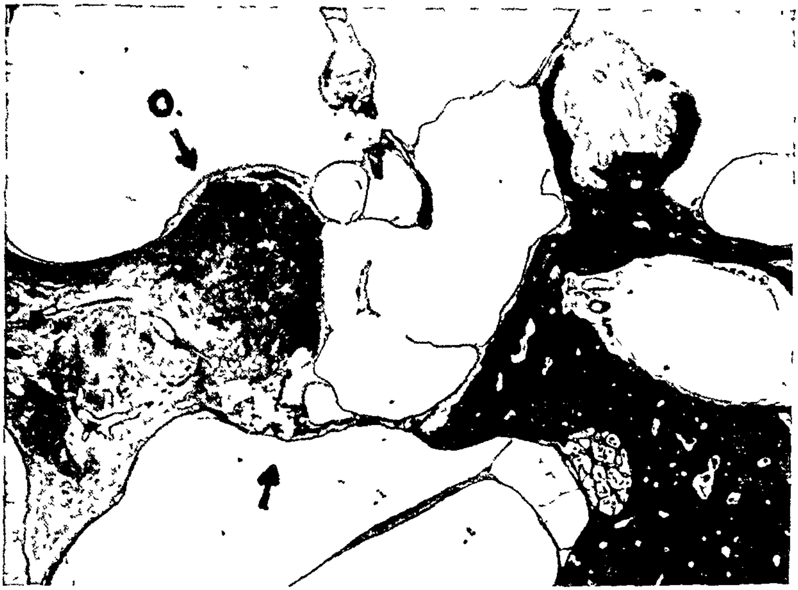

Fig. 2. Section through oval window showing ankylosis by calcification (arrow). After breakage, such calcified spicules would theoretically reform. Note excess fibrous tissue which would be unaffected by manipulation of the head or neck of the stapes.

In Fig. 3 we show a type of otosclerosis where we have extensive new bone growth which involves the entire footplate. I cannot see how anything except breaking of both crura could occur with manipulation of the head of the stapes, in such a case. (At this point I should like to say parenthetically that in histological sections we can show only one part of the curve of each crus in longitudinal section, and, therefore, it is not quite accurate to consider that what you see under a photomicrograph is the entire thickness of the crus. As you know, each crus is hollow with the curve inward. Whether one can mobilize a massive ankylosis of the stapes or not is a great question. Cases of severe deafness having an improvement have been reported. I have been successful in only one case in which I have seen new white bone growing across the entire footplate.

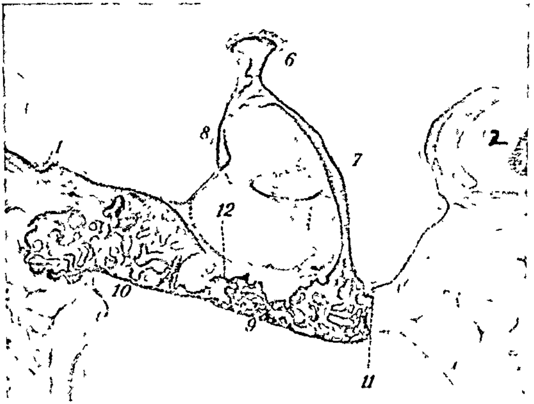

Fig. 3. Nearly horizontal section through otosclerotic stapes With massive ankylosis of this type it is doubtful if mobilization can occur unless there are normal areas in the foottplate which can be fractured. (1) Tympani plexus (2) Facial nerve (6) Head of stapes (7) Posterior crus (8) Upper part of anterior crus (9) Cartilage of footplate replaced by otosclerosis (10) Ankylosed annular ligament (11) Partially ankylosed posterior part of annular ligament (12) Calcified spicule in region of annular ligament (From Bruhl) (1)

Dr. Altmann, Dr. Basek and I found in the laboratory that unless we used the dissecting microscope we could not see what we were doing. In the operating room we cover it with a polyethylene or Dupont Mylar sleeve which is transparent and can be well sterilized (see Fig. 8). We can see exactly what mag-

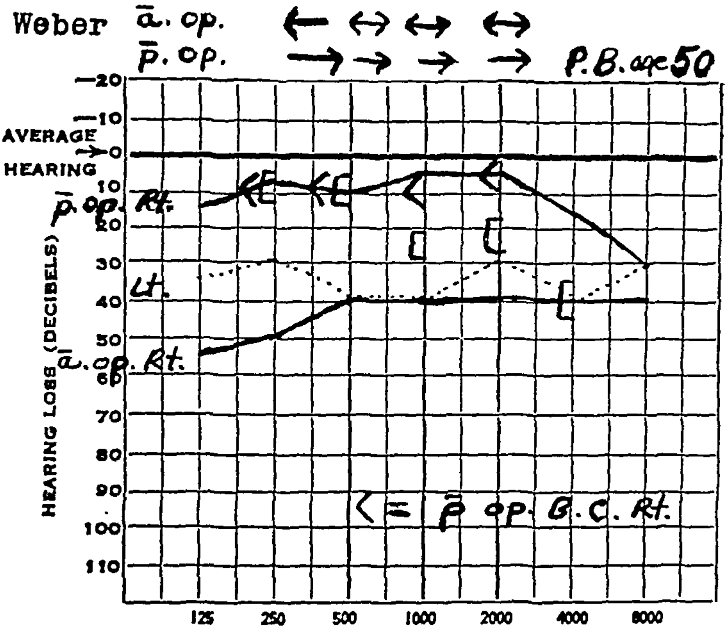

Fig. 4. Audiogram of case of anterior crurotomy, in which inadvertently the posterior crus was cracked, but not displaced. Note elimination of the "Carhart notch" and fact that posterior crus probably rehealed to give such good hearing for air conduction.

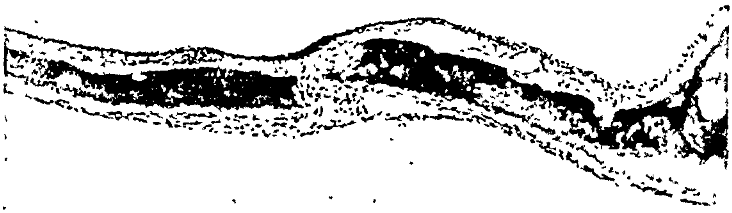

Fig. 5. Healing of fractured footplate by fibrous union. Autopsy 56 years after injury. From Hallpike (2).

nification we are using, with this type of cover, and we can also have the camera in place and manipulate the controls of it without undue difficulty. We work with 4x, 6x and sometimes 10x magnification, but we check the footplate with 16x and 25x, after what seems like a successful manipulation.

I am showing the audiogram in Fig. 5 because I am later showing a movie where the same sort of thing happened as was recorded in the operative dossier of this patient; namely, when the anterior crus was cut, it was seen that the posterior crus was also cracked. There was no displacement of the posterior crus; and I believe that it grew together again, because in the audiogram you will notice that the air conduction improved considerably. Incidentally, the bone conduction postoperatively also improved. The Carhart notch is eliminated, thereby proving the theory of Carhart that a 15-20 db BC loss at 1,000 and 2,000 has something to do with inner ear impedance rather than a loss of nerve function.

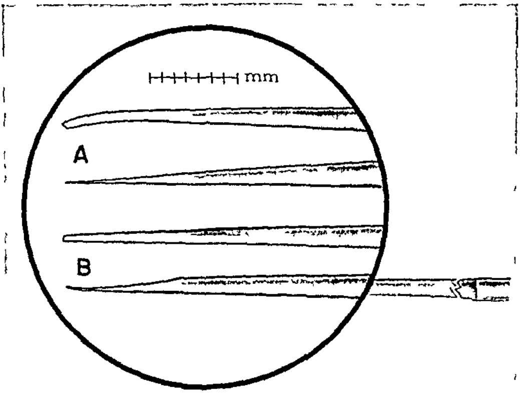

Fig 6 (a) Saber of Basek for weakening and cutting the bone of the footplate (b) Hand chisel of Basek for cutting across the footplate, 0 5 mm width, fashioned from a needle, this is used when the footplate cannot be cracked with a saber Slight pressure and a rocking movement are used

Finally, as we show in Fig. 6, some of our thinking is based on an observation of Hallpike's.[2] This is similar to a case reported by Dr. Altmann[3] where he, too, shows that when the footplate is fractured it does not heal by bony union, just as the fractured endochondral bone of the labyrinth capsule does not heal by bony union. I think that such fibrous union is what we get when we mobilize the footplate by any of the techniques used by the men participating in this Symposium. I do not think that we get any improvement of hearing unless we mobilize the footplate and fracture through its normal areas. I believe we are going to have a good lasting hearing in our successful cases because of this observation of Hallpike, confirmed by Altmann. I also believe that we must design our techniques to produce a fracture of the footplate and to release the anterior crus from the bony growth in the promontory anteriorly.

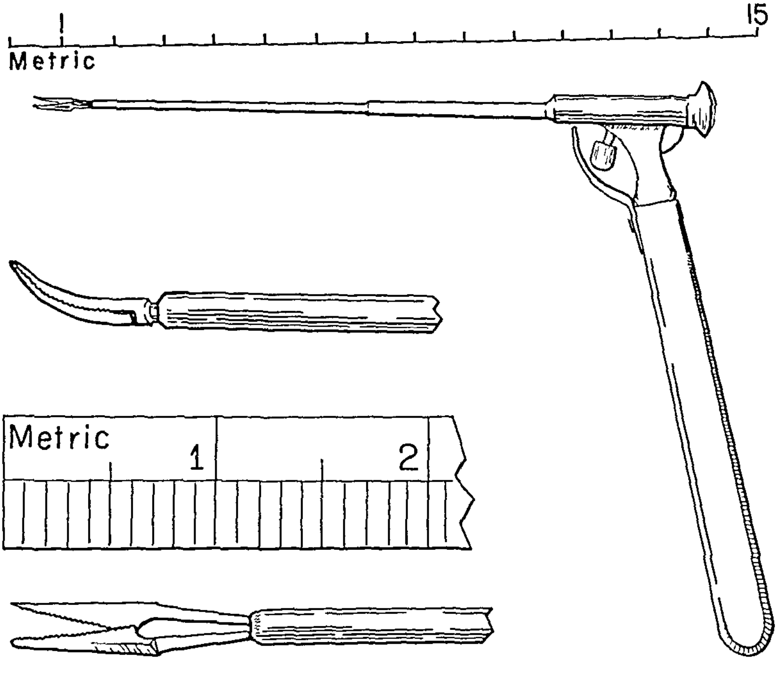

Fig. 7. Anterior crurotomy scissors with correct curve and serrated edges.

That this is feasible was then shown in a movie picture illustrating the following points: The material used was fresh cadaver specimens, artificially ankylosed either with adhesions or with dental wax preparations. It showed the operation as performed by Altmann, Basek and myself as follows: *1.* Incision from above the short process of the malleus through 180 degrees; *2.* Elevation of the sleeve of skin to the edge of the annulus; *3.* Slight inadvertent elevation of the outer epithelial layer of the drum, which was not continued because of the magnification being used; *4.* Entering the Rivinian notch with a pointed needle, so as to perforate the mucous membrane lining of the middle ear and not dissect it away, as sometimes occurs if this maneuver is not carried out; *5.* Elevation of the drum from above downwards, by pressure on the fibrous annulus, thus lifting the drum from the bony annulus from above downwards rather than from outside in; *6.* Cutting away the bone over the chorda tympani to obtain a good view of the footplate and the anterior crus; *7.* Weakening of the footplate with the needle-like sabre of Basek (see Fig. 6); *8.* Cutting the anterior crus with special scissors (see Fig. 7) so that the

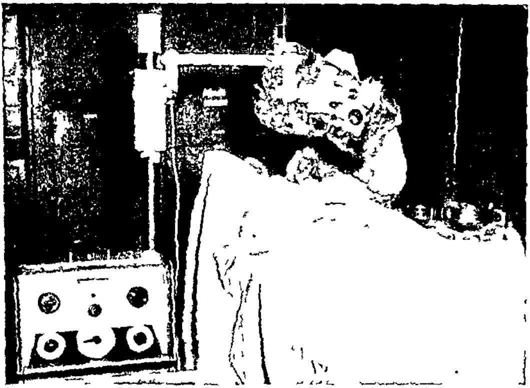

Fig 8 Dissecting microscope (4x to 40x) with multiple movements and counterweighted focus built in illumination 20 cm or 30 cm working distance, flash and camera attached and covered by sterile plastic shield with audiometer on the left Nurse and instrument table in background High rod covered by drapes gives patient comfortable breathing area Objective of microscope is somewhat lower and more tilted when in actual use

shearing action of the scissors crack the footplate in its already weakened line.

The moving picture also showed what occurred with the maneuvers of Rosen; namely, that the anterior crus and the footplate broke if the anterior crus was weaker than the posterior crus when one pushed backward on the neck of the stapes according to the classical method of Rosen. This, however, occurred in only about one-third of the cases. In the rest, both crura broke and the footplate, as a rule, stayed intact. There was some discussion of the normal movements of the stapes, also more detail concerning the anatomical considerations for the anterior crurotomy procedure. Most of this discussion can be found in the June issue of the *Archives of Otolaryngology,* 1956.

In conclusion I acknowledge grants from the Research Council of the American Otological Society and from the New York Foundation. It was these which made possible detailed investigation into the histology of stapes ankylosis and the modus operandi of mobilization procedures.

DR. HOUSE: I believe that the members of our panel have indeed presented to us a very excellent detailed review of what we are doing today in the field of mobilization surgery in this country. I am sure it points out very clearly that there are many problems yet to be surmounted. It is only through the work that you have seen as the result of this symposium, and the work that has been done by many others who are members of this organization, that we can finally evolve some of the answers to the perplexing situation.

I shall make no effort to review each gentleman's presentation; they did it so thoroughly and so beautifully.

I would like to go back a moment, however, and emphasize a few points about indications. I was glad Dr. Fowler emphasized that subject a bit because I too feel that perhaps it was not clearly explained to you gentlemen, many of whom are interested primarily in the indications for this surgery. Many of you will not do this surgery, but you will want to know when it should be done and what to tell your patients.

As far as indications are concerned, I think all of us on this panel are mutually agreed that any patient who is suitable for fenestration is likewise suitable for mobilization. I believe we are safe in saying that every member of the panel will recommend or advocate mobilization first, to be backed up by fenestration if and when it is necessary, and providing, of course, that they are suitable for fenestration. Many patients who are suitable for mobilization, are not suitable for fenestration. One, for instance, the debilitated patient; two, the patient with a unilateral hearing loss who, perhaps, we would not recommend for fenestration but would certainly recommend for mobilization; third, the individual who has a far advanced nerve type of loss, providing there is still a spread between the bone conduction curve and the air conduction curve sufficient to give that individual an improvement, even though he may have to continue with his hearing aid afterward. Nevertheless, such improvement is extremely valuable to many patients. It has been amazing to me, as I am sure it has been to other members of this panel, the difference between having a 60 db loss and a 40 db loss.

Now a word about what is told to patients regarding conservatism, caution and great care in educating them to the overall otologic problem, not with the thought that we are going to do a mobilization and that is the end. We, as otologists, must render to our patients a complete otologic service, of which mobilization is but one part.

As far as technique is concerned, you have heard what is going on in the way of technique. I am sure you will realize there are almost as many different techniques being used as there are men doing the operation. That is to be expected in this relatively early exploratory phase of this new procedure; and, perhaps like fenestration, sooner or later we will evolve a rather constant and standard procedure. You have heard each of us express the fact that it is not a simple operation and is not without possible serious complications. Likewise, it is an extremely delicate procedure, as illustrated so beautifully by our good friend, Dr. Lierle, in his presentation.

With these few comments I will now review the results of the cases forwarded to us for analysis.

In this group of cases (slide) are represented a total of 1,091 mobilization cases operated up to the time of this analysis, one month or longer prior to sending in the information. You will notice that the overall cases total 1,091 and of that number 35 per cent reached the level of 30 db or better. As you will see later, this also includes all of those cases which did not have a chance to reach the 30 db level in some instances, because of rather far advanced nerve deterioration.

We divided these cases (slide) into three main groups, the "A," "B" and "C" cases. We will classify the "A" cases as that group which had a loss by bone conduction of 10 db or less at 500, 1000 and 2000 cycles; therefore the so-called ideal case for fenestration. Of this series of slightly over 1,000 cases, 24 per cent were represented in this group, so that we had 24 per cent of this series that were ideal or "A" cases. At the end of one month the average for all the 1,091 cases which had reached the 30 db level or better was 58 per cent. The highest group was reported as 66 per cent reaching the 30 db or better level at one month. The lowest group in the series was 43 per cent.

We classify as "B" cases (slide) those having a loss of 20 db or less at 500 and 1000 cycles, and 30 db or less at 2000 cycles. This group represented 43 per cent of this series. At the end of one month 38 per cent of these 1,000 cases which fell into this category reached the 30 db level. The highest group reported 46 per cent of these cases having reached the 30 db level, the lowest group reporting 18 per cent.

The "C" cases (slide) are those which have a loss greater than 20 db at 500 and 1000 and 30 db or more at 2000 cycles. The total in this category represented 33 per cent. Those reaching the 30 db level at one month in the average were 25 per cent. The highest group reaching the 30 db level in this group was 54 per cent. That is an interesting figure, because it means that in this particular series it would seem that the bone air gap, the difference between air conduction and bone conduction, would of necessity have to be completely, or almost completely closed, to reach the 30 db level. I would imagine that this is the result of what statistics can do on occasion. The lowest group in this category, 5 per cent, reached the 30 db level in the "C" cases.

Now let us break this down a little further (slide) and see what happened to each one of these operators as a group when we compared their first 25 cases with their last 25 cases. We see that 10 per cent of their first 25 cases closed the bone air gap completely, that is the spread between air conduction and bone conduction. In the last 25, 17 per cent closed their bone air gap completely. How many closed within 10 db of the gap? 13 per cent in the first 25, 15 per cent in the last 25. How many closed within 11 to 20 db of their bone conduction curve? 13 per cent in the first 25 cases, done, in the last 27 per cent; therefore the total, you see, in the first 25 cases of the men in this symposium, 36 per cent did not reach the 30 db level but fell within these three categories, not necessarily being within the 30 db level. The last 25, 59 per cent, fell into these three categories. I think this is extremely significant, because it points out the value of experience in this work, the necessity of the training that must be had, the delicacy of touch, etc., that can be acquired only by working in fixed stapes and footplates.

Now let us see what happened to closure of the bony area (slide). It is interesting to note that 46 per cent of the total cases, therefore, closed the air bone gap within 20 db of the bone conduction curve, regardless of what the bone conduction curve showed.

The average closing to the bone conduction curve completely (slide) was 11 per cent, the highest group 21 per cent; the lowest group 6 per cent; those closing within 10 db of bone conduction averaged 16 per cent; the highest group 37 per cent, the lowest 9 per cent, and within 11 to 20 db of bone conduction the average was 19 per cent; 21, the highest and 10 the lowest.

Now let us talk about our failures (slide); 15 per cent of our cases in this symposium were 0 to 10 db worse by air conduction. That means that if they were even 1 db worse by averages they fell into this category, which obviously is well within audiometric allowances. If we allow 10 per cent averages for the three speech frequencies as being audiometrically questionable, we would say, then, that 3 per cent of our cases were worse between 11 and 20 db by air conduction; so 3 per cent are worse by more than 10 db and less than 20 db, and that 2 per cent were more than 20 db below the preoperative level. If we look at the bone curves, it is rather interesting to note that 4 per cent of the cases operated had a lessening of the bone conduction ability following surgery. They were down 25 db, or less in 4 per cent. Most of those obviously were done relatively early, but this is an interesting and important figure: .2 per cent of the cases were down more than 25 db by bone conduction, thereby indicating apparently a damageed inner ear mechanism.

What about surgical complications (slide)? You heard the chorda tympani problem mentioned by Dr. Shambaugh and others, so we will not include that. We will simply say permanent perforations occurred in .7 per cent. Of the series, continued post-operative infection occurred in .2 per cent; persistent vertigo of an annoying degree occurred in .7 per cent. Facial paralysis, which was temporary, occurred in .3 per cent, probably from infiltration, as mentioned; therefore, the total complications of any practical degree in this case were actually 1.9 per cent. Just a little less than 2 per cent had any type of serious complications actually, except for the two patients with labyrinthine damage. I think that this speaks very well for the fact that serious complications in the hands of those experienced in this work seemed to be relatively few.

By this symposium we have attempted to bring you up to date on our thoughts and views pertaining to mobilization surgery.

DR. HOUSE: I will ask Dr. Rosen to proceed.

DR. SAMUEL ROSEN: It is extraordinary that since my work was first presented at the Triological last year in Hollywood, Fla., some of the most distinguished otologists in this country have reported here their results on hundreds of stapes mobilizations, and I am very pleased that their results tally so closely with my own.

The first patient, a chemical engineer, whose stapes was mobilized in April, 1952, attained normal hearing three weeks after operation and has maintained it ever since.

As for the duration of hearing improvement, out of approximately 1100 stapes mobilizations, all who improved 15 db or more have maintained this improvement for periods up to four years, except for 20 cases which did not fall below their preoperative levels.

I heartily agree with everything that the panelists have said. The presentation of the history of this operation by Dr. Meltzer was excellent. He rather feels, as I know I would if I had always lived in Boston, that that city was really the birthplace of mobilization in this country.

Dr. Lindsay's presentation merely emphasizes what I have felt from the beginning, that most of the time whatever was happening to the footplate during mobilization, I could not see; therefore, while some results turned out good and some did not, I really did not know what I was doing. In my opinion the first real attempt toward solving this particular problem as to what happens at the footplate when we do something at the neck is the way Dr. Ned Fowler is tackling this problem. He makes an assumption which has sense. He says that every time Rosen puts a mobilizer on the neck and successfully mobilizes the stapes so that the hearing improves, he believes that the anterior crus and the footplate are fractured, and he is going to study this problem to see how often this does occur. The fact that it occurs once merely shows that whatever happens at the neck can result in the kind of structural change described by Fowler. Now we have to find out to what degree and how often this really does occur.

Thus far I must say that I paid very little attention to this situation, because it never occurred to me until I met Dr. Fowler and Dr. Altmann a few months ago in Istanbul, where he first presented this idea; at least it was new to me. Ever since then I have in every case tried to observe as carefully as I could, with and without the microscope, to see whether Fowler's idea is correct. I did come across a patient a short time later in whom I could definitely see that I had fractured the anterior crus close to the footplate, but because this patient's hearing did not improve on the table, I assumed that I did not fracture the footplate. I then manipulated the footplate, as I will demonstrate in just a moment, following which the hearing did improve. Maybe I really did an "incomplete Fowler."

In mobilization the variations in the structure of the stapes makes a lot of difference. Anyone who has examined several hundred stapes taken out of cadavers will see the marked changes from one to the other; very frequently from one to the other removed from the same cadaver. I must say, for my own comfort, that when I feel the neck of a stapes, as I told Dr. Day yesterday, I prefer it to be shaped something like his long neck since I am more successful in mobilizing such a stapes than when a stapes has the short neck of a Stewart Nash.

There is no way that I know, as Dr. Kos so correctly said, of predicting whether you are going to be successful in any given case. I have had so many patients in whom I have made a secret prediction that I would or would not succeed, and I can tell you that my predictability is way off. Very frequently I have been unable to mobilize the stapes, where the history of deafness is not more than five years, and I have been able in many cases to mobilize the stapes where the history of deafness is 25 or 30 years. I don't know of any audiological test at present that gives us the key to an accurate prediction.

I think Dr. Goodhill's point about exploratory tympanotomy is a good one. There are times when we are quite uncertain what we will find in the middle ear in conditions other than otosclerosis, and since the lifting up of the drum is such a simple procedure as far as its effect on the patient is concerned, I do not hesitate, though it does not occur very often, to tell the patient, "I do not know what is going on in your middle ear; I can only guess, but I would like to look in there, as a surgeon does an exploratory operation on the abdomen. I may find something which can be helped, but even if I do not find it I do not believe the procedure will hurt you."

Dr. Goodhill has spoken frequently of "incudostapedial lysis." I think we must face the fact that no matter what one calls the operation, if you do not mobilize the footplate you do not get improved hearing. After all, a "Rosen" by any other name smells like a "rose" just the same.

As for Dr. Shambaugh's presentation, his movie was extremely beautiful. I am in the process now of having a film made with Dr. Kobrak at the Otological Laboratory at Wayne University. I have been there three or four times. We have done part of it on the cadaver and part on the living subject. It is very difficult to get your head or fingers out of the way and still see what you are doing. We had to have special instruments made to meet this problem so that I was very clumsy in doing the operation with these new instruments. It is just difficult to make a good movie, but I hope we shall be successful soon.

I think Dr. Shambaugh is correct when he said that we should not have unlimited enthusiasm. I do not know how much is unlimited, but I will bet you that my enthusiasm is greater than his.

As to the operation that I described, I am sure it is not the final operation by any means. I see and hear about modifications that are being made all the time, and I often wonder how stupid I was not to have thought of the things that other fellows are doing with success; but it is going to be a long time, in my opinion, before we can use the distillate of the wisdom of all the various techniques now being tried. I am extremely hopeful.

This (slide) is as I have found it in about 8000 specimens, about half human and half cadaver; about all you see when you first lift up the drum. In 85 per cent of the cases you cannot see more than this much of the incudostapedial joint, and, therefore, I do not believe that this operation can be comfortably and precisely done until you really get a good exposure, as shown in the next slide

The difference between trying to do a mobilization with th slide just removed as compared to this one (slide) is as th one were trying to do a tonsillectomy in a child with its mouth half open. With this exposure I feel comfortable. I can see the structures. I can see the relationship of the structures to other structures, and one thing that I believe is very important in this operation is for one to be as relaxed all over as possible. If you do the operation sitting down you should be leaning on the patient, with (they don't seem to object to it) your arm on the patient. You should have a footstool under your feet. You should not go ahead with the mobilization when your feet are tense. If you are tense elsewhere, and you think you are relaxed in the muscles directly concerned with mobilization, I do believe that one is apt not to do it so well as if one is relaxed all over. There are times during the operation when I find myself becoming tense and when I do I just come out, put the instrument down until I relax the tensions. Most people think I am looking for another instrument. I am just

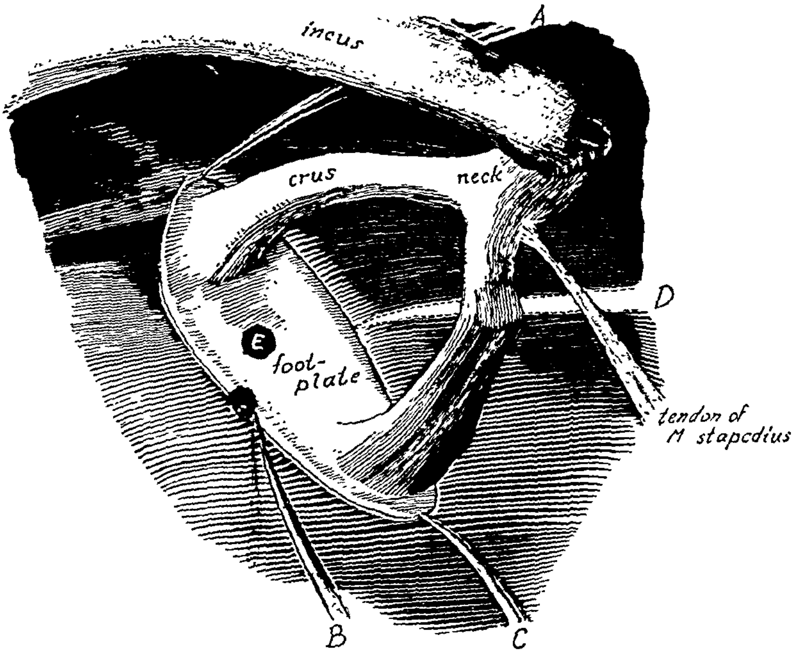

Fig. 1. (E) indicates the site of the fenestra ovalis through the body of the footplate.

waiting for my blood pressure and pulse to go down a bit, then I pick up the instrument and try to start in again where I left off.

This (slide) brings up a very important aspect in the development of stapes mobilization. There is a good percentage of patients who, when you try mobilization at the neck, just will not mobilize. You will either quit because the crura are so thick that nothing will happen, the patient's hearing will not improve, or as more often happens one or both crura are fractured. In the early days when I felt that I was licked at this point I quit, but as time went along I decided to try very gingerly to introduce an exploratory needle between the bony ridge of the oval window and periphery of the footplate itself for a distance not greater than the thickness of the stapedial footplate, to pry the footplate loose. To my amazement I could sometimes see that as I introduced the instrument here and

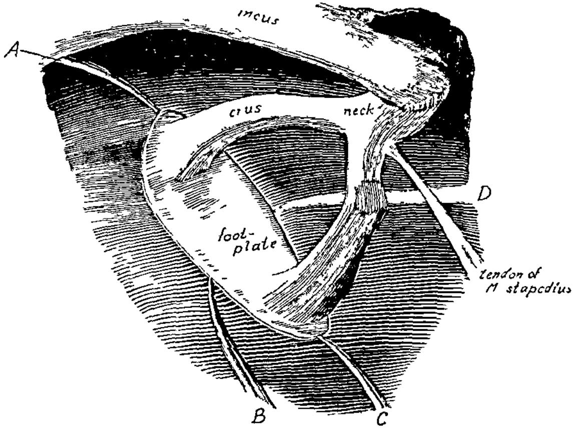

Fig 2 "A" and 'B" show the pointed explorer used anteriorly and inferiorly to mobilize the footplate Occasionally the explorer is used at the posterior edge of the footplate "C' and rarely at the anterior edge "D" just tried to move it or pry it a little, I could move it. I must say that when I first started doing this, my heart was in my mouth every time I tried it. This feeling still persists, although to lesser extent; however, I do recommend that in cases where the attempt to mobilize at the neck fails for one reason or another, we should not abandon the mobilization at that point. I might add that in some of the patients in whom I was able to mobilize the footplate itself successfully the hearing improved to levels as high as those in whom I had successfully mobilized at the neck, even though in some of these cases I could see that the crura were completely fractured. I do not quite understand this; but I know that it has happened, and I just mention it for your information.

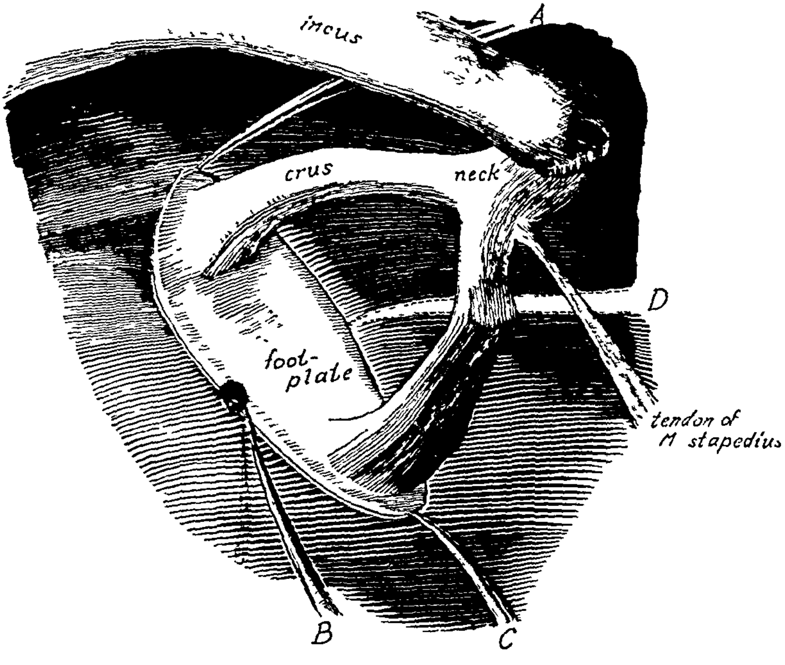

Fig. 3. Fenestra ovalis at the inferior margin of the footplate created by penetrating the explorer (B) for ½ mm into the vestibule leaving a circular opening about half the size of the head of a pin, after attempts to mobilize the footplate at A, C, and D failed.

In this picture (slide) I have had a few patients in whom I could not move the footplate at any point. I decided that I

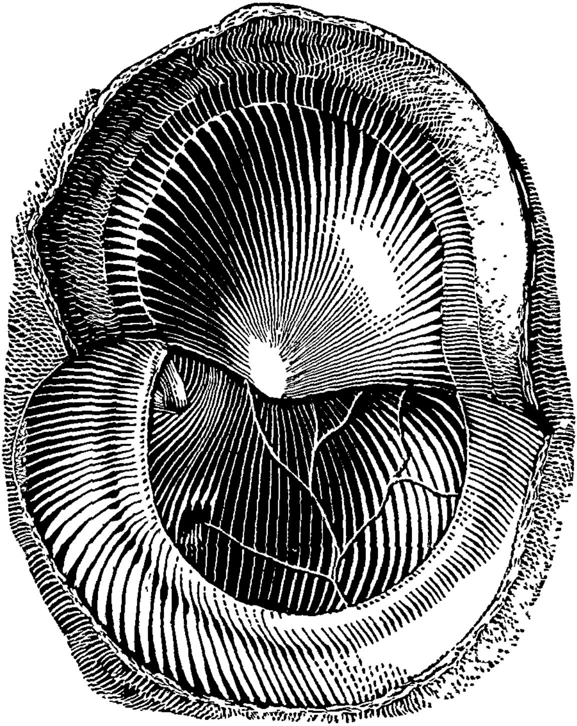

Fig 4 When drum and skin are reflected upward, in over 85 per cent of the cases more or less of the incudo-stapedial joint is seen This is, however, an inadequate view of the stapes that does not allow safe manipulation

would try to make an opening into the vestibule which would include a small portion into the footplate itself, and again this was done both in the presence of crura which could not be mobilized at all and also crura which had already been fractured. In these patients—and this could really be called a

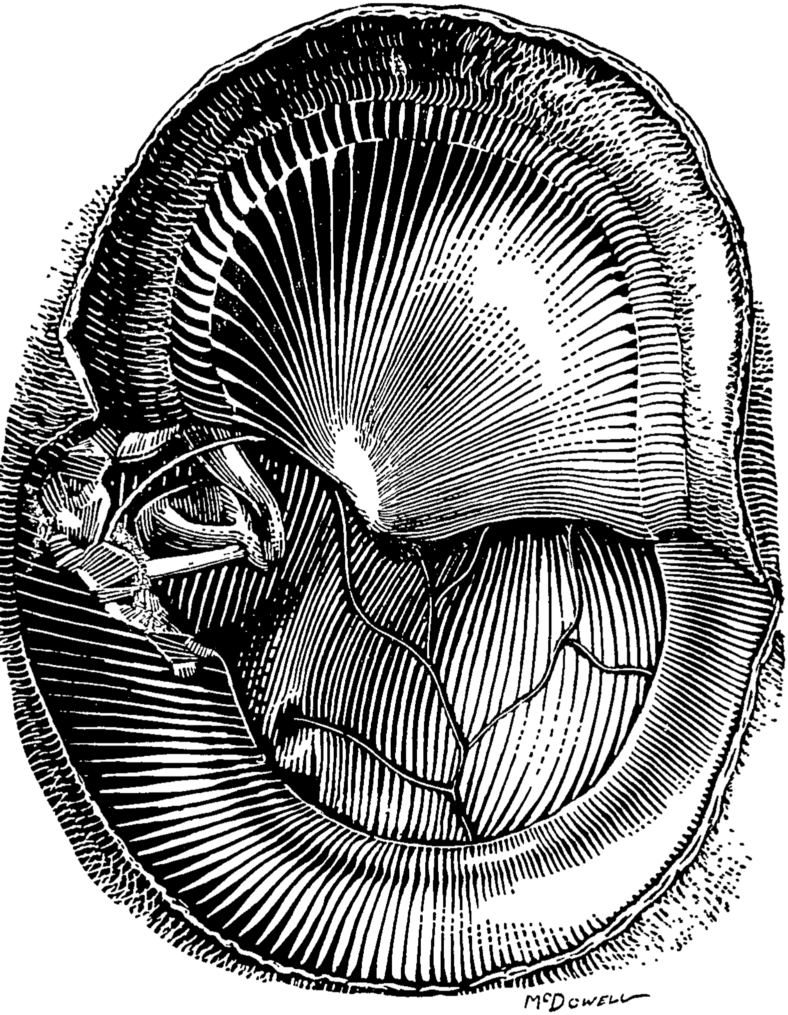

Fig. 5. After 2-3 mm. of the very edge of the posterior bony canal just external to the incus and stapes is removed, a full view of the long process of the incus, the incudo-stapedial junction, the head, neck of the stapes and the entire length of the stapedial tendon, etc., is obtained. This exposure is absolutely necessary for stapes mobilization.

fenestration of the oval window or vestibulotomy—the hearing improved to extremely high levels. I suppose that one could explain this in the absence of the ossicular chainn, by the fact that somehow the organ of Corti is stimulated, and I suppose the organ of Corti does not give a hoot how it is stimulated so long as it receives stimulation.

This (slide) demonstrates some of the achievements with this operation at the neck or at the footplate itself. We have had normal hearing, and almost everyone who is doing mobilization has also achieved normal hearing. Some have had normal hearing restored in both ears. There are some patients in whom the bone conduction will not permit normal hearing, but you can still close the gap and they obtain excellent hearing, even though the bone conduction is not normal.

Restoration of useful binaural hearing is one aspect of this audiological relationship to which we do not pay too much attention. It is quite amazing the difference there is between binaural hearing and monaural hearing. You can test this for yourselves by placing your finger in one ear canal and see what it sounds like. After you have held it there for a minute see what it sounds like when you hear in both ears. You find that the difference is enormous. If the patient has excellent hearing in one ear and relatively poor hearing in the other ear, to mobilize the second ear in order to achieve binaural hearing I believe is a worthy goal.

The fourth step is to remove the stapedial component where there is a marked mixture with nerve deafness. The fifth is to restore enough hearing to allow the successful use of a hearing aid in extreme cases with deafness.

I also had a movie, but I think you get the point that I am trying to make. The purpose of this is merely to show the importance of getting the proper exposure; without a wide exposure it is extremely difficult if not impossible to do this operation. Here (film) the exposure shows the incus, the stapes, the stapedial tendon. This was done on a patient operated upon in Dr. Croushore's service at the Receiving [illegible]pital in Detroit. The most important things to see [illegible] footplate, the incus, the incudostapedial joint. My [illegible] this surgery, no matter what technique is employed [illegible] the exposure ample by removing enough of the [illegible]tory canal edge

Now a word or two about the revisions. I have done 96 revisions in patients who had no improvement after the initial operation; 25 per cent of those operated a second time reached a level of 30 db or better, and 40 per cent showed improvement though they did not reach the 30 db level.

Recently I operated upon a series of 77 cases in which the footplate could not be moved at all, or in which the crura were fractured. Previously, as I said, I abandoned these patients because I knew of nothing more to do; however, since I have been employing the footplate technique, not only have I revised those patients on whom I previously failed, but also in whom at the initial operation on these 77 cases I was unsuccessful in mobilization and which would have been abandoned, I went immediately to the footplate at the initial operation. 34 per cent of these patients showed improvement where they reached 30 db or better, and 54 per cent showed improvement in hearing but did not reach the 30 db level. These patients previously would have been failures, and a good percentage of them now can be improved.

I have estimated that approximately 5000 stapes mobilizations have been done in this country up to the present time. I have been all over Europe and the Middle East teaching this operation, and I believe that about 2000 of these operations have been done abroad.

It was Stewart Nash who made it possible for me to present this work in the first place over a year ago, so in the end, it is really Dr. Nash who is responsible for having brought these thousands, and I hope tens of thousands, out of a lonely world of silence into the world of hearing.

I am very grateful to Dr. Joseph Goldman, the chief of my service at Mount Sinai Hospital. He is an excellent scientist, a very shy fellow, but he has been right behind me on this work from the very beginning and encouraged me a great deal. I also thank Dr. Moe Bergman in charge of the audiology who worked very hard to keep it on the spot.

DR. BARRY J. ANSON: Emphasis on three structural features of the stapes is a warranted addendum to our previous presentation: the degree to which the ossicle has been reduced in bulk during the fetal period; the unusual weakness of the basal extremity of each crus; and (so far as known) the inability of the stapes to heal in an area of fracture.

Were there time, the discussion could be extended to include a review of the process of internal "remodeling" which the incus undergoes throughout the adult life span, and to a degree which varies with the individual. These changes, while different from those which take place in the development of the stapes, have a similarly weakening effect.

Both processes were described in a paper read at the meeting of the American Otological Society in Boston in 1954.

DR. HOUSE: I thank you for your contribution to the anatomy of this area. Since Dr. Rosen mentioned the experiences he is having with permanency over a period of time with his results, we should also make a statement about permanency in our group, although no reported cases in our series exceeded six months. We limited it to six months after surgery, and that hurts. To begin with it is interestting to note that 2 per cent of the cases that improved at the time of surgery and improved subsequently for a week or two, receded to the preoperative level within the first month. Beyond that, little occurred in recession of hearing loss until the fourth month, when 2 per cent closed and returned to their preoperative level, only 9.100 of 1 per cent, and 9.100 of 1 per cent the second month, that is,in the fifth and sixth month of each six months, 9.100 of 1 per cent closed in each month, so that the percentage, as near as we can analyze the trend at the moment at least, is that most of them are going to close up within the first month. If they bridge that period they will go along pretty well to their fourth month, when they will again tend to heal over, and beyond that it looks as though there is less likelihood of closure occurring.

DR. FRANZ ALTMANN: I will just emphasize one point that Dr. Fowler discussed, *i.e.*, that no hearing improvement is, of course, possible unless there is a mobilization of the footplate. This mobilization is often achieved by manipulation

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DR. BARRY J. ANSON: Emphasis on three structural features of the stapes is a warranted addendum to our previous pre-here today. We have been using a very conservative technique and have experienced very few complications as a result of the mobilization of the stapes.

If we consider operating upon the footplate of the stapes we should attempt it as a primary procedure, so that the ossicular chain is not disturbed in any way. If we disturb the ossicular chain and go to the footplate after the crura of the stapes are fractured, then we will not obtain the maximum of improvement possible.

So far I believe we do not possess a technique which is safe to use at the footplate of the stapes. The footplate is in a very protected position, and the ossicles above it make the surgical approach very difficult.

We are in the process of developing an instrument which we hope will allow for better visualization and manipulation of the footplate. Until a more satisfactory technique is available for the surgery of the footplate I shall approach this phase of the work very carefully.

DR. HOUSE: Dr. Bellucci, I thank you, and will now ask Dr. Shea to comment.

JOHN J. SHEA, JR.: In reading the original literature on the subject of mobilization, my interest was aroused by a case reported by Jack of Boston in 1902.* As Dr. Meltzer pointed out, these original pioneers proceeded from where we are now in mobilization of the stapes to removal of it. This case was reported with good hearing ten years after bilateral removal of the stapes. There had been an adhesion between the eardrum and the oval window. It was reported that the patient could hear a soft spoken voice at 20 feet. That captured my imagination. I attempted to elevate the canal flap in such a way as to drop the drum on the oval window, but I could not do it, although I tried many times; also, in creating a long enough cuff I was afraid that if I left the drum down on the exposed either on the neck of the stapes or the head of the stapes or the lowest part of the incus; in other cases, however, it is necessary to do manipulations on the footplate. These procedures are, however, not without risk. This was shown by a case which I operated a few months ago: on the table, after fracturing the footplate with the pneumatic hammer, an improvement of 35 to 40 db throughout the range with almost complete closure of bone air conduction gap was achieved. Although the patient was kept under antibiotics (achromycin 250 mgm. three times a day) he nevertheless developed a labyrinthitis on the sixth day and ended up with a completely dead labyrinth. This shows that we should give thought to the following: Are we justified in attempting to get a higher percentage of good results by increasing the danger to the patient? I think we are justified since stapes mobilization is still in the experimental stage. We will never be able to make real progress unless we take certain risks.

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* Jack, F. L: Supplementary report on a case of double stapedectomy operated upon 10 years ago. Tr. Am Otol Soc., 8.99, 1902.

There is one more point. I was very interested in the improvement in hearing in cases where both crura were completely fractured. Our group in Presbyterian Hospital has given a great deal of thought to this; we feel it is difficult to understand how this could occur if both windows would vibrate in the same phase. Sometimes the fracture in the posterior crus might heal, particularly when no dislocation had occurred. In other instances blood clots might become organized in the round window niche; in this way the phase difference between the two windows could at least partly be restored. We even thought about reviving the old Hughson procedure, in such cases, and put a piece of muscle into the niche to give some sound protection to the round window.

DR. HOUSE: Dr. Bellucci will now give us his comments.

DR. RICHARD J. BELLUCCI: Before this wonderful symposium I thought I had something to say. I now find I cannot add much to the discussion. We have done a large series of cases at the Manhattan Eye, Ear and Throat Hospital and we have achieved 30 per cent good results in an unclassified group of patients. This is comparable to a similar group presented put an eye in the top of it to receive the end of the incus, to re-establish connection between the stapes and incus.

Fig. 2 shows the view from the top showing connection between the stapes and incus. In this case I was able to lay the artificial stapes right over the oval window.

I did this operation only several days before I left to come to the meeting, so I cannot report on the exact outcome. This is a completely preliminary report. Postoperatively, the woman was able to hear very well in the operated ear.

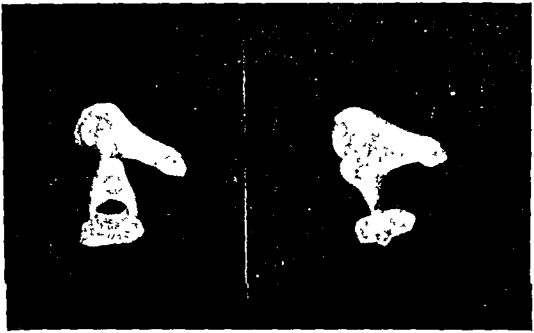

Fig. 2.

I have not tested her audiometrically. She had no useful hearing in that ear preoperatively, and recently her husband told me she was able to hear the gong of a church bell a quarter of a mile away, which she had not heard since she was a little girl.

DR. HOUSE: Thank you. These are all the variations of technique that are in the minds of many people regarding this operation. I should now like to ask Dr. Baron to discuss this symposium.

DR. SHIRLEY H. BARON: I should like to bring out just one point that has not been mentioned. It has been said that fenes-oval window the patient might blow his nose and push it off. That was my first effort.

In addition I was very hesitant to do anything too vigorous about the footplate. It seemed to me most unreasonable to tamper with it or tap on it because of the ease with which it could be knocked loose. It was then that I decided that these two ideas could be combined. I decided to fenestrate the oval window. I selected a patient for this procedure on whom I had already done a successful mobilization of the other ear. She had a very marked hearing loss in this ear, down about 75 db for air and about 55 for bone. She was bothered by a blowing tinnitus. I exposed the bone in the usual way, attempted to mobilize it and I could not. I then removed the stapes and covered the oval window with a very thin skin graft, approximately .005 inch thick. I replaced the stapes with an artificial one I had created of nylon, from detailed studies of about 100 specimens. This artificial stapes was designed by a mechanical engineer who studied the stress problem involved, and strangely enough created one much like the original.

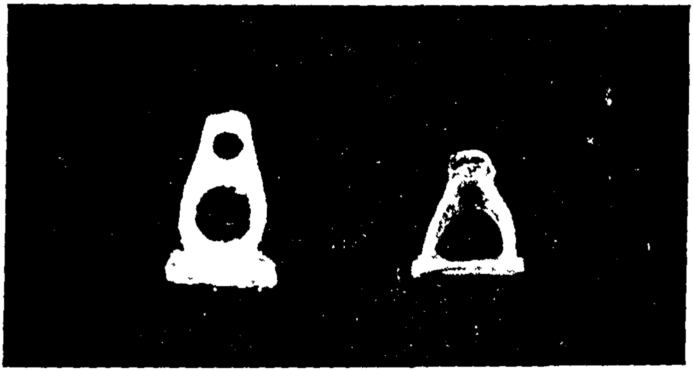

Fig. 1.

In Fig. 1 you can see the stapes I used. The human stapes from the base to the top measures about .140 inch. The one we created is the same height from the footplate to head. I

1955. The black line is her preoperative air conduction level. At surgery I fractured the crura, so I mobilized the footplate. The audiogram at surgery immediately following this, as noted by the green line, showed a very poor result. It looked

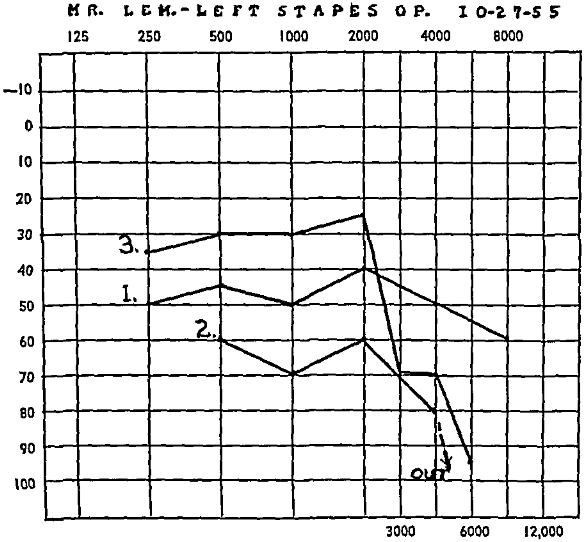

Fig 2 1 Preoperative Audiogram, 2 Postoperative Audiogram at surgery. Direct manipulation of stapedial footplate after crura were fractured Note that frequencies above 4,000 are out, 3 Audiogram two months after surgery Some improvement in frequencies from 250 to 2,000, but no recovery in high tones

as though the hearing had been made worse; however, the actual postoperative result, shown by the red line, is excellent. The air-bone gap has almost been eliminated. This improved threshold was reached within two weeks after surgery and has been maintained ever since. The evidence here is clear that one cannot always tell at the time of surgery whether or not tration can successfully follow mobilization, and that we know. It is not advisable, however, to fenestrate at the same operation should the mobilization appear to be successful. I present a case to show why one should defer the fenestration until a later date, in the hope that this may stop the tendency to do both procedures "in one sitting" as is currently the practice of a few otologists.

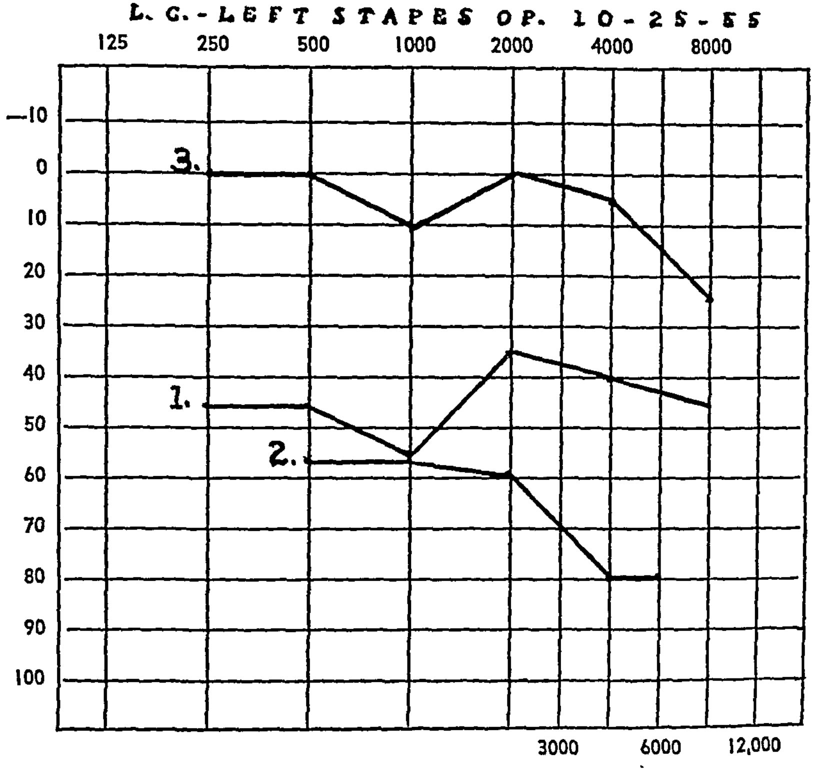

Fig. 1. 1. Preoperative Audiogram; 2. Postoperative Audiogram at surgery, following direct manipulation of stapedial footplate after crura had been fractured. Hearing appears to have been made worse, but recovery was spectacular as noted in audiogram 3, six weeks later. Conclusion: One should not fenestrate at the same operation if mobilization seems to have failed.

(Slide) This case proves that one cannot always tell at the time of surgery whether or not the attempted mobilization will fail. This is a 53-year-old female operated upon in October,

1955. The black line is her preoperative air conduction level. At surgery I fractured the crura, so I mobilized the footplate. The audiogram at surgery immediately following this, as noted by the green line, showed a very poor result. It looked as though the hearing had been made worse; however, the actual postoperative result, shown by the red line, is excellent. The air-bone gap has almost been eliminated. This improved threshold was reached within two weeks after surgery and has been maintained ever since. The evidence here is clear that one cannot always tell at the time of surgery whether or not

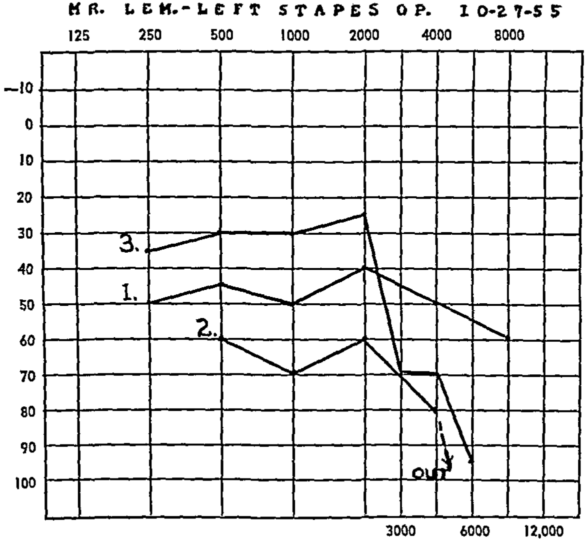

Fig. 2 1 Preoperative Audiogram; 2 Postoperative Audiogram at surgery. Direct manipulation of stapedial footplate after crura were fractured. Note that frequencies above 1,000 are out; 3 Audiogram two months after surgery. Some improvement in frequencies from 250 to 2,000, but no recovery in high tones the mobilization is successful; and that fenestration should be deferred until a proper conclusion can be reached.

(Slide) While the former case shows what can be done beneficially as the result of mobilizing the footplate directly, the slide of this case shows what might happen adversely. The black line shows the preoperative level. I fractured the crura, and then mobilized the footplate directly. An immediate marked hearing loss, especially for the high tones, as shown by the green line, resulted. This patient became very dizzy right after surgery and stayed dizzy for about four days. The red line shows that, postoperatively, the hearing did improve in the conversational range but not in the high tones, which became worse. Incidentally, the patient remained dizzy for about six weeks. The result in this case suggests that the traumatic force applied to the footplate might be transmitted to the basilar membrane in the first turn of the cochlea.

J. H. THOMAS RAMBO, M.D., New York, N. Y.: I had not planned to enter the discussion, but since the general idea of applying the drum membrane directly to the oval window after removal of the stapes has been brought up it is perhaps proper, and even necessary, that I make a preliminary report of a surgical technique directed to this end which we are in the process of testing, both clinically and in the animal laboratory at the New York University-Bellevue Medical Center. We are also studying the effects of this procedure on the inner ear in monkeys.

This technique differs from that which has just been described by Dr. Shea in that it does not use any free skin grafts or any prosthetic devices. It consists of an approach to the middle ear in the same manner as that described by Lempert and now used in stapes mobilization with the difference that initial incisions are made at the junction of the outer third and the inner two-thirds of the external canal so that the reflected canal skin from the posterior canal wall is longer. After reflection of the skin flap and the drum, an area of bone is removed at the Rivinian notch as in mobilization. The incus is then removed. With the drum folded forward one can obtain a very good exposure of the long handle of the malleus.

With a small golf-club spud, used to remove foreign bodies of the eye, the inner layer of the tympanic membrane surrounding the malleus handle is incised, and the malleus is separated from the drum. I first did this in 1953 in performing a modified radical mastoidectomy, as an aid to cleaning out pathology in the middle ear without destroying the ossicular chain. Since that time I have done this on a number of occasions in the operating room and many times on the cadaver. After a little practice it is surprisingly easy to do, without tearing the drum. After removal of the incus and the malleus the mucous membrane in the vicinity of the oval window is scraped away. When all bleeding is controlled and the middle ear is dry, the stapes is then removed from the oval window. The drum membrane, or adjacent flap skin, is placed over the oval window, invaginated, and the invaginated area packed externally with a small plug of cotton in the manner as described by Lempert in the fenestration operation. The skin flap attached to the drum is then replaced against the bony posterior canal wall to close the middle ear.

We then have a system for mobilization of the inner ear fluids which is the same as that following a fenestration operation except that we have substituted the oval window for a surgically made fenestra in the horizontal semicircular canal. The drum becomes plastered to the promontory, but there remains an air-filled tympanic space in the hypotympanum with free communication between the round window and the Eustachian orifice. We do not, of course, retain the advantage of sound transmission through the ossicular chain, but you have all heard the remarkable results given this morning, without explanation, when simple perforation or fracture of the footplate has been accomplished following fracture of both crura. Dr. Rosen has reported revisions in previous failures which yielded 34 per cent results of 30 db or better when a footplate approach was used. I believe that experience with the footplate approach in mobilization techniques is leading us inevitably to the recognition that hearing through even a small opening at the oval window is a much more physiological way for airborne sound to mobilize inner ear fluids; and we are, therefore, getting surprising good results when sound reaches the inner ear through this natural route, even without an ossicular chain.

As I have observed these fracture techniques of the footplate, I have gradually overcome what has, for several years, been a stumbling block in my mind to the development of a predictable procedure for permanent hearing gain following stapedectomy. This has been the difficulty of removing a really fixed stapes. With the use of the percussion hammer, however, when it is necessary, I cannot now foresee any fixed stapes that could not be fractured and the two or more pieces hooked and pulled outward without injury to the inner ear. The advantage gained by separating the malleus from the drum, which can then be reflected forward to expose the entire middle ear, also allows infinitely better exposure for accomplishing this at the recessed stapes area.

In this symposium today we have been reminded by several of the participants that mobilization is not a standard procedure; that there are many techniques used; and that we are all looking for a special procedure which will offer a better solution to this surgery. I imagine that approximately the same situation probably existed following Miot's experiences with mobilization which prompted Jack in Boston, after repeating them, to perform stapedectomy. If such be the case, that we continue to follow faithfully the experiences of the otologists of over half a century ago, which we have done so far, we will again arrive full-circle to stapedectomy. Jack, however, was unable to find a way of closing the inner ear to obtain a successful hearing result and, because of the hazards of infection, abandoned the procedure. The technique which I have described may furnish the answer to what has been missing—a successful way to close the inner ear, while maintaining a functional tympanic space, following stapedectomy. I believe this will be the next logical step forward in otosclerotic surgery. Such a technique offers the same advantage as the fenestration operation in that it is a definite and predictable procedure. It offers the advantage of the mobilization approach in that it avoids a mastoidectomy and the problem of postoperative care. It, furthermore, assures a permanent hearing result in that it avoids the principal causes for postoperative hearing loss of both operations. Since mobilization of inner ear fluids is through nature's own window, it will not close, and since the stapes footplate is removed, reankylosis of the footplate cannot be a factor after this procedure.

Dr. Lewis F. Morrison: First of all to qualify myself, I do not do the fenestration operation. Secondly, I am in a teaching institution, and I have a certain amount of responsibility in regard to what may or may not be done by the members of the staff. Thirdly, I think I share with you a responsibility to the public. I would like either your assent or negation of the statement I often have to make that it is not correct for an individual who is not qualified to follow through with the fenestration procedure to do a stapes mobilization operation. If I am correct I would like to have some acclaim. If I am incorrect, please correct me.

Dr. House: Thank you, Dr. Morrison. I am sure that this certainly does reflect the entire attitude of our panel. As I mentioned in the summary, I feel that this work is only a part of an otologic service that should be rendered to your patient; therefore, unless you are thoroughly familiar with temporal bone anatomy, thoroughly qualified in temporal bone surgery, I do not believe that you should undertake to render only a partial service.

If there is no other urgent discussion, I again turn the meeting back to our President, Dr. Lierle.

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President Lierle: I cannot close this meeting without saying that I have known that this presentation has taken months of good hard work, and I congratulate Dr. House and his panel on this most excellent presentation. I think it is one of the best we have had. I also thank the people of Montreal, including the men here, their wives and all the contributors who made this program possible.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

Washington, D. C., May 5-10, 1957.

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DEVELOPMENT AND ADULT ANATOMY OF THE AUDITORY OSSICLES IN RELATION TO THE OPERATION FOR MOBILIZATION OF THE STAPES IN OTOSCLEROTIC DEAFNESS *†

BARRY J ANSON, Ph D , Med Sc (By invitation),
Chicago, Ill ,
and
THEODORE H BAST, Ph D , Med Sc (By invitation),
Madison, Wis

I INTRODUCTION

It is a privilege to present those aspects of our study of the stapes which may be serviceable to the participants in the later symposium

This report represents one phase of an inclusive, long term investigation conducted under the auspices of the Central Bureau of Research of the American Otological Society We take special satisfaction in the invitation of your President, Dr Dean M Lierle, to appear on this program, whereon laboratory research and clinical practice are to enjoy logical and helpful interrelationship

Our concern now is with the structure of the stapes insofar as its architecture affects technical procedures calculated to mobilize the innermost member of the ossicular chain, ankylosed by spread of sclerotic bone into the normal space of the vestibular fenestra (oval window) and against the base (footplate) of the stapes

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* Read at the Sixtieth Annual Meeting of the American Rhinological Laryngological and Otological Society Montreal Canada May 15 1956

† Contribution from the Department of Anatomy Northwestern University Medical School (Contribution 624) and from the Department of Anatomy University of Wisconsin

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It is a striking fact that even a relatively sturdy stapes is still a congenital weakling, a veritable wraith when compared to its earlier self in the midterm fetus. The normal steps in development account for stapedial fragility; they are basic to whatever may be said about adult structure.

It is fair to regard the stapes as a "nonconformist among bones." The incus might justifiably be placed in the same category, but for different reasons; in fact, the three ossicles originate in an exceptional way, continue on a course of morphogenesis which is not only noncompliant, but also precipitate, to attain adulthood before the infant is born.

II. OBSERVATIONS AND DISCUSSION.

Even as early as the second month of intrauterine life (8½ weeks, 28 mm.), the future ossicles are already formed in cartilage, distinguishable from the elements of the branchial skeleton from which they were derived, in relation to the mandible.*

This means that in the course of their morphogenesis as cartilaginous, and later as osseous skeletal elements in vertebrates, the ossicles must undergo a remarkable transformation. Their predecessors functioned as supports for the respiratory mechanism in aquatic and amphibious vertebrates; in mammals they are made over to serve as links in an ossicular chain.

A. *Developmental Stages Shown by Reconstructions.*

When first distinguishable, in the fetus of eight weeks, the "stapes" is not yet stirrup-shaped (see Fig. 1-a);** rather, it is a ring.

Within the short period of a week (see Fig. 1-b), the characteristic portions are identifiable, but more prominently still (see Fig. 1-c) one week later (11 weeks, 78 mm.) and in slightly older specimens (see Figs. 1-c and 1-d).

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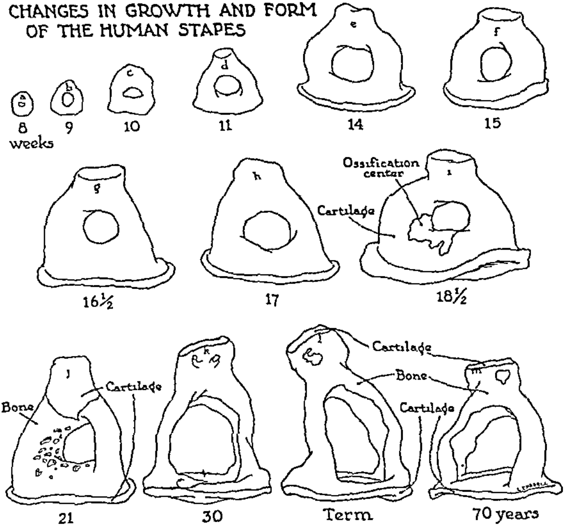
* See Fig. 144, the present authors' monograph, in "The Temporal Bone and the Ear" (C. C. Thomas, Springfield, 1949).

** Figs. 1-a to 1-m are used here in place of several lantern slides which accompanied the presentation of the paper; the remaining figures (2-a to 2-f, 3-a to 3-d, 4-a to 4-f, 5-a to 5-c) were prepared as contact prints of lantern-slide negatives actually shown during the reading of the paper.

At the close of another period of growth, three weeks in length, the ossicle is a stirrup (see Fig. 1-e). Soon the base of the stapes becomes lipped; the head is foveate to accommodate the lenticular process of the incus (see Figs. 1-f to 1-h).



Figs 1-a to 1-m Drawings of reconstructions of stapes. Specimens selected to demonstrate eleven successive stages in morphogenesis (*fetal stage indicated in weeks*) and to show variability in form and size of mature ossicles in the fetus at term and in an adult of advanced age (From Anson and Cauldwell: Quarterly Bulletin of Northwestern University Medical School, 15, 263-269, 1941)

In the 18-week fetus ossification has begun, in a solitary center on the obturator surface of the base (see Fig. 1-i).

The progress of ossification is rapid. Within a period of three weeks, bone has spread along the crura; upon reaching the neck of the ossicle, bone has become continuous circumferentially. Growth thereupon ceases (see Fig. 1-j). Ossification involves excavation, a process which serves to alter profoundly the structure of the ossicle.

In the stapes of the fetus of 30 weeks, periosteal bone has been removed on the obturator surface of the base, in the crura and neck (see Fig. 1-k). As a result, the stapes has been hollowed throughout. Cartilage now remains only on the articular surface of the ossicle and on the vestibular and fenestral aspects of the base. The addition of endochondral bone has converted the base and the head into two-layered plates.

At term (see Fig. 1-l) and in the adult (see Fig. 1-*m*) cartilage persists on the vestibular (medial) surface of the base and on the fenestral margin as a peripheral rim. The tympanic (lateral) surface, on the contrary, is formed by a plate of irregular bone which is composed of both endochondral and perichondral tissue. The crura are deeply channelled. The head and neck of the ossicle are strikingly eroded; the capital extremity, like the basal end, is bilaminar.

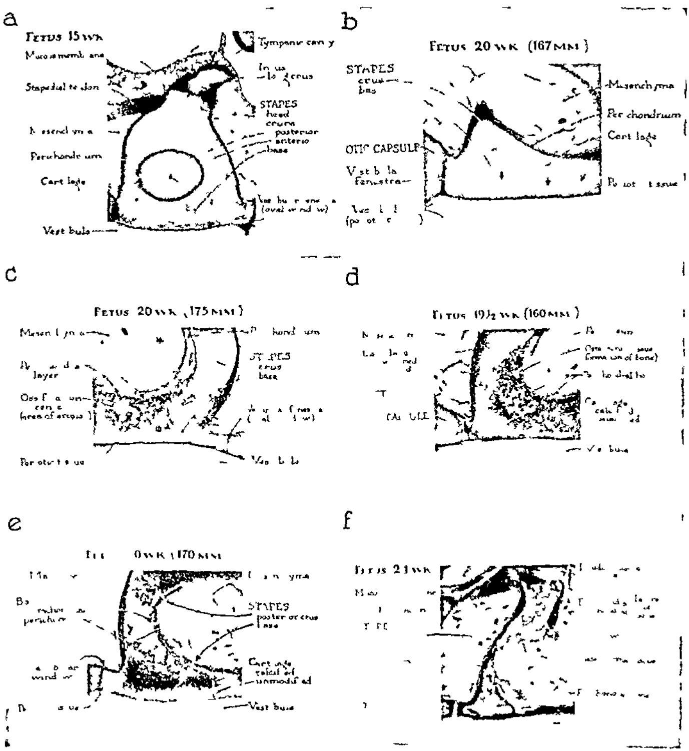
B. *Developmental Stages Shown by Sections.*

Reconstructions, like the 13 whose changing structure has been reviewed, demonstrate the "gross" fate of the originally bulky ossicle. They require supplement, through the use of photomicrographs, to account for the histological processes involved in the transformation.

Near the beginning of the fourth month (112 mm.) of intra-uterine life, the future ossicle has acquired characteristic form; however, it is composed wholly of cartilage (see Fig. 2-a).

In the stapes of the 20-week (167-mm.) fetus, ossification is merely predicted in the vacuolization of the cartilage on the tympanic (or obturator) aspect of the base (see Fig. 2-b). The altered cartilage is distinctive, the line of separation from the unmodified portion (indicated by arrows) being that which subsequently divides an osseous from a cartilaginous layer on the stapedial base.

In another specimen of approximately the same stage, and to the depth previously indicated, bone-formation extends in

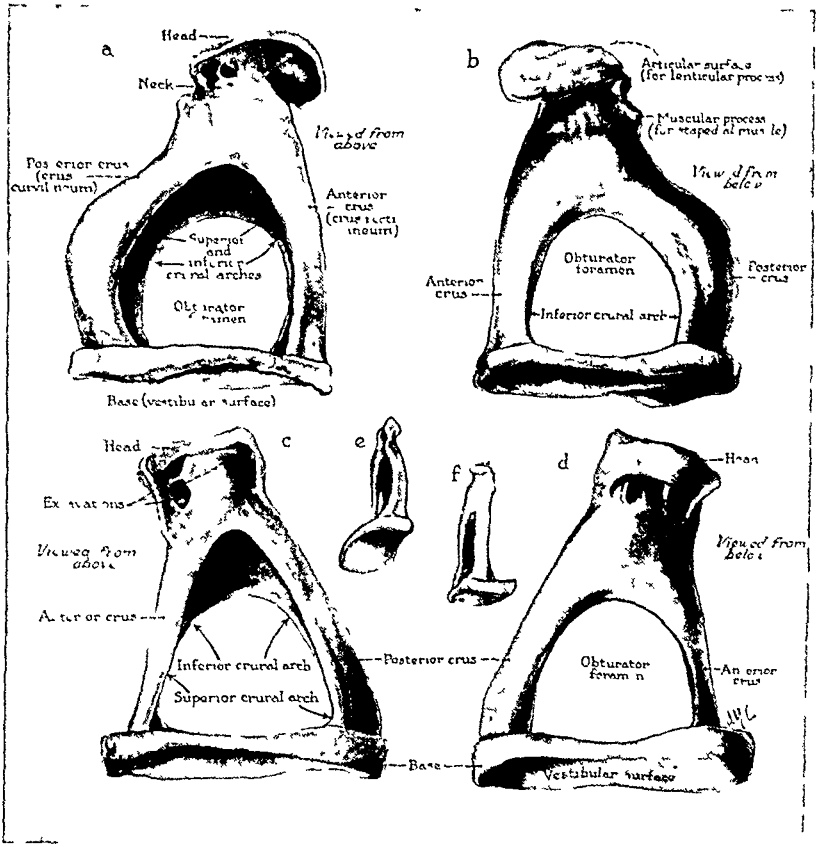


Figs 2 a to 2 f Photomicrographs of transverse sections through the stapes Showing the primordial histological structure of the stirrup and steps in the conversion to bone Fig 2-a entire ossicle Fig 2-c anterior crus and base others posterior crus and base

the solitary ossification center (see Fig. 2-c). Perichondral bone is being produced on the surface, while erosion is occurring at deeper level.

Removal of cartilage is now a striking feature (see Fig

2-d). Heralding the advance of osteogenic tissue, the cartilage becomes calcified. Perichondral bone, as a layer, is incomplete, being destroyed as it is freshly formed.



Figs 3-a to 3-d Photomicrographs, continued Fetal, infantile and adult structure of the ossicle

Where the process of ossification is furthest advanced (mid-length of the crus), perichondral bone forms a complete shell (see Fig. 2-e). Endochondral bone, transitorily formed, has been removed, and is replaced by marrow. The crura are now hollowed columns of perichondral bone, which contain mere remnants of old cartilage and newly-formed marrow.

In a specimen of 24 weeks (see Fig. 2-f) the obturator wall is a mere remnant (resorbed between the marking arrows). Marrow, now openly exposed to the mesenchymal tissue, will be replaced by the submucosal layer.

The full effect of this developmental process is demonstrated in the fetus of four and one-half months. Throughout the length of the crus, the mucous membrane occupies the former marrow-space of the ossicle, resting against the surface of the crus which was once internal.

Endochondral bone, newly-formed in apposition with the cartilage of the base of the stapes (see Figs. 3-a and 3-b), spreads across the tympanic aspect of the footplate (in the direction of the unlabelled arrow, in Fig. 3-b), finally covering the eroded surface of the cartilage. Although the addition of this new layer sometimes virtually doubles the thickness of the base, the latter is still far thinner than it was in the four-month fetal stage. The bone thus produced is not solid; neither is it evenly spread through its extent. Actually, the accumulation is thick circumferentially and is quite highly vascularized (see Figs. 3-c and 3-d). Numerous intercommunicating channels occur; the contained blood vessels are branches of those in the submucosal tissue.

As a result of the action of the processes whose progress has just been traced, the stapes in the ear of the infant (see Fig. 3-c), and therefore, the ossicle of the adult (see Fig. 3-d), bear little resemblance to their ring-shaped forerunner in the eight-week fetus.*

C. *Adult Form, Demonstrated by Specimens.*

It now remains to account for the adult form and size of the stapes. This will be done through the use of excised and "cleaned" specimens, drawn by the artist—as the ossicles were seen through a high-power binocular microscope. Four selected specimens will suffice for an introductory description.

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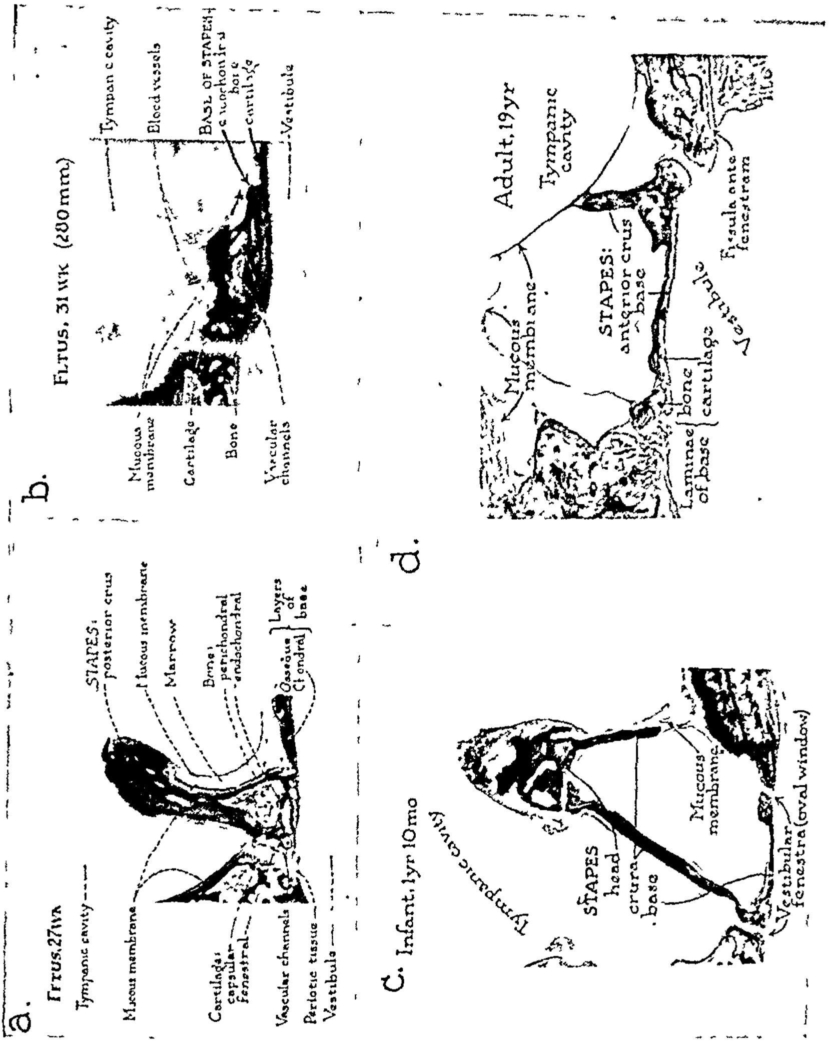
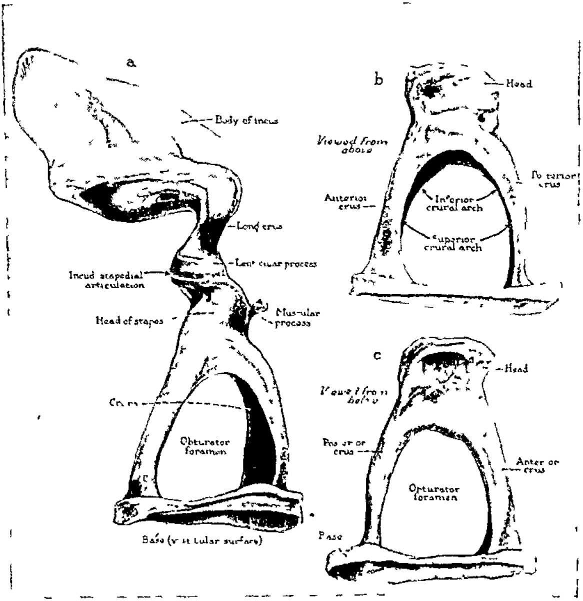
* Detailed and amply illustrated accounts of the morphogenesis of the auditory ossicles may be found in the following journal articles by the present authors and in related publications cited therein Annals of Otology, Rhinology and Laryngology Vol 55 pp 467-494 1946, and Vol 63, pp 394-434, 1954; Quarterly Bulletin of Northwestern University Medical School Vol 28, pp 17-45, 1954



Figs 4-a to 4-f. Drawings of excised, "cleaned" specimens of adult stapes (Figs. 4-a and 4-b, Specimen I; Figs. 4-c to 4-f, Specimen II).

In Specimen I the head of the stapes is deeply pitted, as is often the case. The head, relatively large, is inclined anteriorly (see Figs. 4-a and 4-b). The muscular process is fairly prominent; it is attached to the neck (as in 75 per cent of cases), not to the head proper. The superior crural arch (toward the observer in Fig. 4-a) is more expansive than the inferior (viewed in Fig. 4-b); it is roughly triangular, whereas the inferior one is ovoid. Here, as usual, the anterior crus is the more slender of the two and the straighter (hence the term, *crus rectilineum*). The posterior crus is curved in this instance, as it most frequently is (so, the term *crus curvilineum*).



Figs 5-a to 5-c Drawings of specimens, continued Fig 5-a, Specimen III, Figs 5-b and 5-c, Specimen IV

In Specimen II the head meets the crura in an even curve. Like the preceding example, the head is deeply pitted (see Figs. 4-c and 4-d). The articular surface is foveate. The crura, deeply channelled and having crural arches of different dimensions, are less sturdy than those of the other specimens. The posterior crus is almost straight. Both the posterior crus (toward the observer in Fig. 4-e) and the anterior crus (facing the observer in Fig. 4-f) originate from points nearer the inferior than the superior margin of the base. In this specimen the crura meet the base at almost a right angle; however, this angle of junction may be acute, either superiorly or inferiorly.

In Specimen III a prominent muscular process is present; it arises from the neck, rather than from the head (see Fig. 5-a).

In Specimen IV the head is large and quadrilateral in outline; it is inclined anteriorly to a very slight degree (see Figs. 5-b and 5-c). The head is sulcate across, but not pitted. The crura are almost equal in respect to curvature (and, therefore, length) and to strength. As is almost invariably the case, the superior crural arch is greater in circumference than the inferior.

The outcome of these processes is the presence, in the human ear, of a bone whose average weight is less than three mgms. (2.86 in 75 specimens, to be exact), and whose adult form would suggest profound skeletal emaciation. To make matters worse (from the viewpoint of the otological surgeon), the ossicle is weakest where we would like to find it strong, that is, at the basal extremity of each crus.

III. SUMMARIZING STATEMENTS.

The stapes is the oddest bone in the human body. Salvaged, as it were, from the remains of a respiratory mechanism functional in aquatic and amphibious creatures, this segment of the branchial skeleton undergoes dramatically rapid transformation to become a stirrup-shaped ossicle of full adult dimensions just as the human fetus passes through the middle month of its intrauterine life. The stapes is as mature several months before the infant is born as a tibia (for example) is in a person 20 or more years of age; the stapes lacks the usual mechanisms which cause lengthening and thickening in a typical long bone of the human skeleton.

Histologically, the constituent bone of the stapes is primitive; it is never replaced by tissue of Haversian type.

The transformation which converts the crura into channelled columns, the head into an excavated cylinder with a bilaminar articular extremity, and the base into a thin, two-layered plate results in striking reduction in bulk. The several parts are excavated, and the space thus exposed is made continuous circumferentially to face the obturator foramen—the latter space being greatly enlarged at the expense of that which originally contained marrow and endochondral bone. The surface of bone thus exposed by erosion is draped by mucous membrane; blood vessels in a submucosal position communicate with others which course peripherally in the endosteal bone of the head and of the base.

In cases of otosclerosis, with threatened ankylosis of the stapes, the features just described assume critical significance. Circumstances of this kind, dependent, as has been demonstrated, upon normal morphogenesis of the stapes, are basic to the challenge which confronts the otologic surgeon in the effort to restore mobility of the stapes.

Reduced to the status of a skeletal ghost, the stapes, in the otosclerotic patient, awaits the inexorable advance of pathological bone from the wall of the window in which it rests—with inescapable and deforming ankylosis.

STRUCTURES OF THE SPIRAL PROMINENCE AND EXTERNAL SULCUS AND THEIR RELATION TO THE ORGAN OF CORTI.*†

MERLE LAWRENCE, Ph.D. (By invitation),

Ann Arbor, Mich.

Much of the confusion that presently exists in the interpretation of the physiological activities of the inner ear is the result of insufficient information concerning the morphology of the structures involved. The truth is that all the facts are not yet in, and since the number of theories seems to vary inversely with the number of established facts, we are today confronted with a myriad of possibilities concerning the activities within the cochlear duct.

Many "explanations" of the mechanism behind pathologic processes within the inner ear are based upon notions of endolymph flow and constitution, and the structures on the "lateral"‡ wall of the cochlear duct, whether absorbing or secreting, are always assumed to play a part. Such hypotheses will remain purely speculative until they are founded on more exact knowledge of the anatomy found within the scala media.

This paper describes structures within the region of the *prominentia spiralis* which have not previously been reported; structures which may play a very important, if not the major, role in the provision of nutrient material to the cells of the organ of Corti.

The present day conception of this region stems from the report by Shambaugh[1] in 1908: a description of the anatomy of the external sulcus in which he observed a small tubule sug-

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* Read at the Sixtieth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Montreal, Can., May 15-17, 1956.

† From the Physiological Acoustics Laboratory, Institute of Industrial Health, and Department of Otolaryngology, University of Michigan. This research was supported in part by the Research and Development Division, Office of The Surgeon General, Department of the Army, under Contract No. DA-49-007-MD-634.

‡ This use of the term lateral refers to a direction away from the modiolus: part of the time this would be medial with respect to the skull.

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gesting a secretory function of the cells. Subsequently this has been called Shambaugh's gland. To the time of the discovery of this tubule the establishment of a secretory function for this area has had a turbulent history, but as is usual, attempts to settle a theoretical argument led to more accurate observation and the elucidation of morphological facts.

The problem really starts in 1836 with the publication by Breschet,[2] a monumental work in which he describes the substance which is inserted into the spiral groove of the bony capsule. Upon dissection this appears as a gelatinous tissue connected between the bony wall and the basilar membrane which Breschet called "the yellow band," or "membranous belt."

A decade later Todd and Bowman,[3] in a textbook filled with original notions, decided that this structure to which the basilar membrane was attached was a muscle that might serve the purpose of accommodation similar to that of the eye, but they felt that, more than likely, "this very interesting structure— [is] placed there to defend the cochlear nerves from undue vibrations of sound."

Then followed a period of the most careful dissection and observation with low magnification, the high point of which was the classic description by Corti[4] of the organ which now bears his name. One of the earliest observers was Kölliker[5] who described in some detail the cells that made up this "cochlearis muscle" of Todd and Bowman, and pointed out that no "Faserzellen" as seen in elements of involuntary muscle could be found. Corti agreed with this, but described another type of cell that was different from the connective tissue cells. He left the question open as to whether or not the structure was a muscle, and used Kölliker's name for it, *ligamentum spirale*.

Claudius[6] described the cells lying along the scala media side of the basilar membrane, a layer of cells extending from the tall mound of Hensen cells on the "lateral" end of the organ of Corti to the concave region between this layer and the spiral prominence. The cells, which had puzzled Corti, were found by Deiters[7] to lie in this concavity of the spiral ligament, and

STRUCTURES OF THE SPIRAL PROMINENCE AND EXTERNAL SULCUS AND THEIR RELATION TO THE ORGAN OF CORTI.[*][†]

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There have been many descriptions of these cells since, but the main difference among observers has been in the interpretation of their function. Boettcher did not specifically say that the spiral ligament was a muscle, but he did say that it was conceivable that these cells of the external sulcus could have contractile properties operating through fibers, which he described earlier,[9] lying above Claudius cells and connected to the reticular membrane. On the basis of this he was described, by Shambaugh, as supporting Todd and Bowman in the muscle theory; but in a paper, 18 years later[10] Boettcher vehemently denied ever having been in favor of the idea of a cochlear muscle, and had only mentioned the possibility of these cells having contractile properties as a speculation, but offered no evidence.* Actually Todd and Bowman, and later Boettcher, were referring to different structures; the former were referring to the entire area of what we now call the spiral ligament, while Boettcher referred to the row of external sulcus cells which had not been described at the time of Todd and Bowman.

Following the careful description of these cells by Boettcher there were several more (Gottstein,[11] Prenant,[12] Katz,[13] Retzius,[14] and others), some backing the thought that the cells possessed contractile properties and others emphasizing that there was no real evidence for this view. Gottstein offered the opinion that these cells represent a neuroepithelium, but could find no nerve fibers connecting with them.

This brings us up to the description given by Shambaugh[1] which has been accepted as the final word for almost 47 years. He studied the labyrinth of the domestic pig, and stained by Mallory's method. This showed the group of epithelial cells that occupy the deep part of the *sulcus externus*, clearly distinguishing them from the adjacent cells.

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* I am indebted to Dr. Stacy R. Guild for having called my attention to this later article by Boettcher

As the earlier observers had noted, these cells send long finger-like processes deep into the spiral ligament. The cells do not form a continuous band spiraling around the turns of the cochlea but appear in a clustered arrangement. Between these clusters the lining epithelium exists as a simple layer of cuboidal cells similar to those which cover the surface of the spiral prominence. Occasionally there is a cavity at the open surface of the external sulcus where many epithelial cells penetrate the spiral ligament. This shows up most clearly when the section is made at right angles to the modiolus. It is quite possible that Boettcher[8] also saw these, but thought that they were openings left when he pulled the finger-like cells from their recesses, which some of them probably were. Within this epithelial bundle there frequently appears a small but clearly defined tubule. This is very fine and is located in the center of the epithelial cell-filled cavity. In the pig these could be found only in the basal turn of the cochlea.

Because of the presence of this tubule and the extreme vascularity of this region, Shambaugh designated this area as the principal source of endolymph and later[15] suggested that the stria vascularis (generally accepted since Corti as the source of endolymph) is merely a mechanism for producing pulsation in the endolymph bringing about the "continuous flow of tonus impulses to the skeletal muscles which clearly emanate from the cristae."

The observations reported here are only of the spiral prominence and external sulcus, and came about by chance when a silver stain being used with celloidin-embedded sections for the study of nerve fibers turned out to reveal the fibers of the basilar membrane, and the cells of the external sulcus and the spiral prominence.

MATERIAL AND METHOD.

During the course of a study carried out by Dr. J. H. T. Rambo at the Lempert Institute of Otology in New York and sponsored by the Lempert Research Foundation, several Rhesus monkeys were perfused intravitally with Heidenhain Susa fixative. The temporal bones were removed, processed, embedded in celloidin and sectioned. A number of random sections were made available to us for experimenting with various silver stains.

The stain used on the material presented here is a modification of the Bodian silver method. Since all the staining was experimental no two sections received exactly the same treatment. The main variations consisted of the time they were left in the protargol solution, the time left in the gold chloride solution, if used, and an occasional substitution of analine oil for oxalic acid. Actually, none of these variations seemed to change the picture to any great extent, the manner in which the tissue takes the stain having already been determined by the acid in the fixative. If a fixative containing no acid is used this stain is specific for nerve fibers and cell nuclei, but when acid is used in the fixative, all tissues are stained, particularly the fibers of the basilar membrane.

OBSERVATIONS.

A clear distinction must be made between the cells of Claudius and those of the external sulcus. The Claudius cells are cuboidal or sometimes columnar. They are uniform, distinctly shaped, and clearly nucleated, resting upon the fibers of the basilar membrane and extending from the tall columnar Hensen cells on the "lateral" side of the organ of Corti to a region beneath the bulging spiral prominence. At this point the external sulcus cells are encountered, and with most stains are clearly distinguished from the Claudius cells. These cells of the external sulcus not only take a deeper stain but they also have long slender processes that extend well beyond the basilar membrane fibers which serve as a base for the Claudius cells. These processes, however, cannot always be seen with conventional stains, but become easily perceptible when a silver stain following an acidified fixative is used*; also with this treatment many other features are revealed.

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* Retzius[14] was the first to demonstrate these by means of a silver stain

Fig 1 The external sulcus cells and spiral prominence as seen in the Rhesus monkey The cells of the external sulcus show long processes that extend between and around capillaries within the spiral prominence A bundle of fibers from the basilar membrane branches off to form a hand-like grip on special cells (see Fig 4) in the lower outer portion of the bulge Monkey 142R 277 x 470

Figure 1 shows the root-like processes of the external sulcus cells as they extend into the spiral prominence The early observers, particularly Retzius[11] described these processes as extending back into the spiral ligament Prenant[12] had also suggested that it was not improbable that the processes of one cell anastomosed with those of another, but Retzius could find

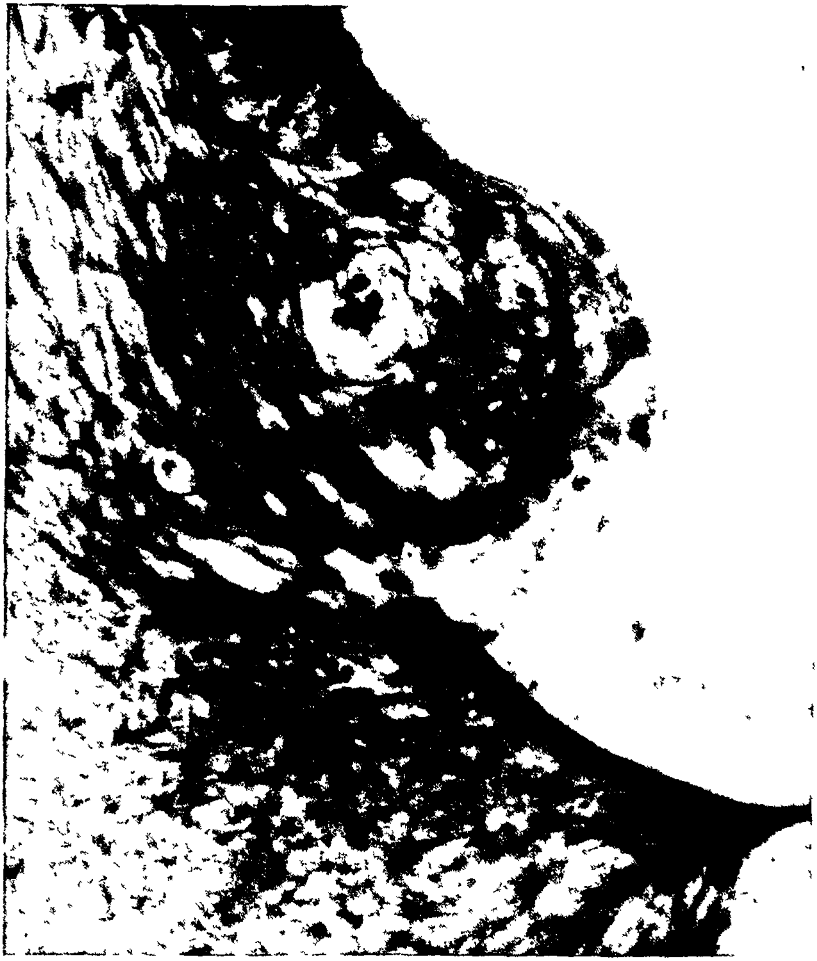

Fig. 2. The opening from the external sulcus into the region behind the spiral prominence. These lie between the bundles of basilar membrane fibers shown in the previous figure, and are filled with cells. It is through these cells that Shambaugh's tubule passes There is an *indication of* such a structure in this section, but it does not show up in the reproduction. Monkey 141L-222 x 490.

no evidence for this. It was also stated (Retzius) that the nuclei lie well back in the region of the spiral ligament.

In the material examined here these particular cells extend their processes into the spiral prominence rather than the spiral ligament. The distinction between the two is not hard to make when the continuing fibers of the basilar membrane are stained as distinctly as in these sections. It is also apparent that there is no observable anastomosis between cell processes, and that the nuclei lie well toward the external end. The openings which these processes appear to go between and around (see Fig. 1) are small capillaries. In describing the position of the nuclei and the direction of extension of the processes the earlier anatomists must have confused these cells with the epithelial cells that Shambaugh described as containing a tubule. There are openings occurring every now and then which extend in channels under the spiral prominence and back into the spiral ligament. These appear filled with epithelial cells as described by Shambaugh and shown in Fig. 2. There are, then, two distinct types of cells which alternate in occurrence around the spiral of the sulcus. It is in the midst of these epithelial cells that Shambaugh saw the tubule. Although our material has not revealed a tubule as clear cut as that of Shambaugh, who resorted to sections horizontal to the coil of the cochlea rather than vertical to it (parallel to the modiolus) the section of Fig. 2 shows an indentation and line between cells which closely resembles the diagram published by Shambaugh.

In this photomicrograph (see Fig. 2), below the opening in the sulcus, a capillary large enough to allow only the passage of a single red cell at a time can be seen. In the middle of the spiral prominence is a larger vessel cut in cross section and containing several red cells.

As noted in all previous investigations this region is highly vascular, containing many capillaries of various sizes. Fig. 3 shows a large branching capillary extending from the bulging region of the spiral prominence deep into the spiral ligament with another branch reaching up toward the region beneath the stria vascularis. Another is seen below the point where the main branch of fibers separates from the basilar membrane to extend into the spiral prominence.

If a section is made at right angles to the modiolus so the cut is made through the bulge of the spiral prominence, or occasionally with a cut parallel to the modiolus, the hook of the basal turn twists so that the prominence is cut essentially in the same way, the relation between the capillaries and the

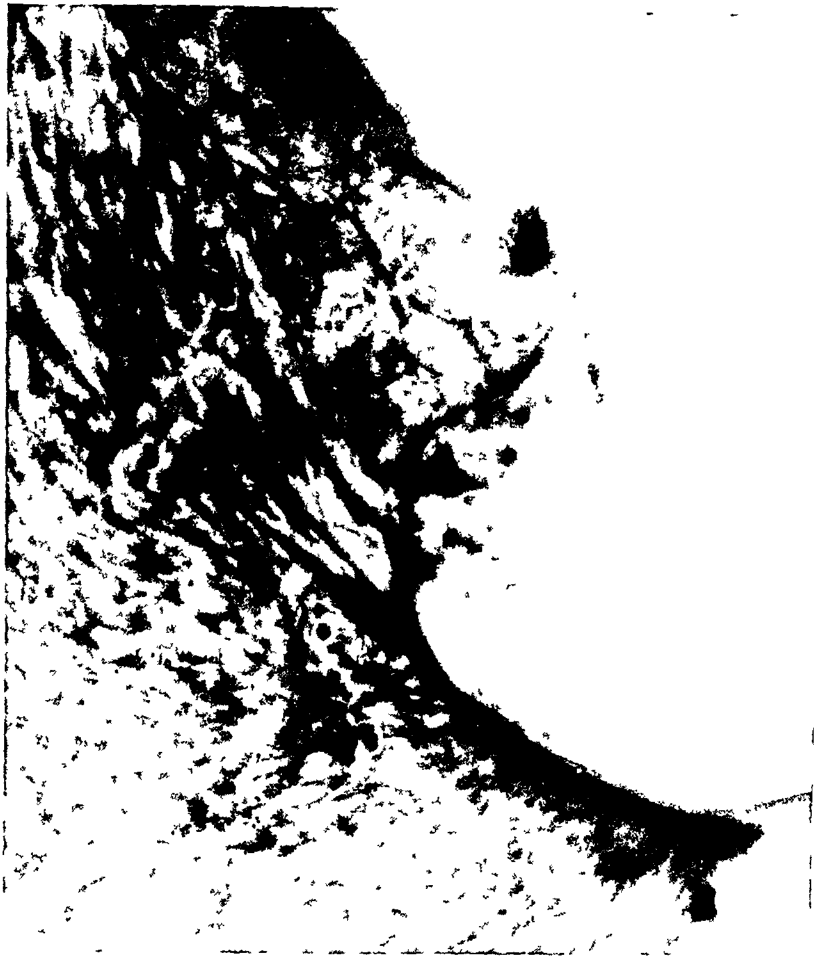

Fig 3 A picture of the spiral prominence to show the vascularity of the area and the infiltration by the fibers from the basilar membrane The capillaries can be distinguished by the presence of many red cells Monkey 141L-225 x 530

"root-cells" can be determined. These capillaries spiral around with the prominence but undergo much looping and twisting; at the same time they go over and under the processes of the "root-cells" much in the manner of the fibers of a woven mat.

The fibers of the basilar membrane as they split up just behind the external sulcus are distinctly revealed in these sections. In Fig. 1 the basilar membrane is the dark line curving

Fig 4 Here is shown the branching bundle of fibers from the basilar membrane going to the group of cells which appear clustered in the form of a rosette The external sulcus cells and their nuclei can be clearly distinguished from the Claudius cells Monkey 142L 331 x 475

up from the lower left Upon reaching the region of the external sulcus cells there is a distinct branching of the fibers One branch extends into the structures of the spiral prominence and the other breaks up into many more, extending up and out into the connective tissue of the spiral ligament These latter fibers are very extensive, some going behind the stria vascularis and extending to its other end The branch to the spiral prominence is of particular interest. It divides quite profusely and appears as a complicated snarl around the capillaries and cells of the prominence. A regularly occurring termination of the basilar membrane fibers appears as a hand-like structure in the lower portion of the bulging prominence. Within this meshwork a cell nucleus can be seen.

A clearer picture of the true nature of this structure can be seen in Fig. 4. These fine fibrils from the basilar membrane intertwine among a rosette of cells. There appear to be 10 to 12 cells in this rosette which, since they do not appear in every section, probably do not continue as a tube spiraling around the turns of the cochlea but occur rather periodically. In this section the type of external sulcus cell that has the processes can be distinguished clearly from the Claudius cells and the nuclei of several "rosette" cells stand out distinctly. The branch of basilar membrane fibers extending to and invading the "rosette" cells can also be seen. We have seen these cells in the basilar turn only.

This branching of basilar membrane fibers does not occur as a continuous band around the cochlea behind the external sulcus. The branches are in groups like fingers on a hand but unite again as they approach the clusters of "rosette" cells. Within the regions where these fibers separate the second type of external sulcus cells (epithelial cells) appear and in sections through this region (see Fig. 2) one sees a picture almost identical with the drawings presented by Shambaugh.

DISCUSSION.

Shambaugh's observations are completely supported by the material used in this study. In addition there have been revealed within the spiral prominence, clusters of cells appearing in the form of a rosette infiltrated by fibers which show a direct continuation with the basilar membrane. There is evidence here to suggest a functional relationship between the basilar membrane and the structures of the spiral prominence.

It is conceivable that the "rosette" cells provide a needed nutrient material which is released or forced out when the interwoven fibers from the basilar membrane are put into action by vibrations entering the cochlear fluids and moving the basilar membrane. The observations of Crowe, Guild and Polvogt[16] are quite significant in this connection. They found a deterioration of the external sulcus cells whenever there appeared a deterioration of the organ of Corti. Specifically, they never observed a deterioration of the organ of Corti without deterioration of the external sulcus cells in the same area, but in some instances the deterioration of the external sulcus cells extended beyond that of the organ of Corti. This led them to suggest that the external sulcus cells play a role in the providing of nutrient material to the cells of the organ of Corti and that if these failed the organ of Corti in that region deteriorated; failure of function of the external sulcus cells occurs first. The intimate relation between the fibers of the basilar membrane and the structures of the spiral prominence suggest that the more active a particular region of the basilar membrane, the more nutrient material forced from the "rosette" cells. Perhaps the stria vascularis merely provides a constant supply of endolymph to serve as a medium for the special nutrient material.

There is still no obvious explanation of the function of the cells with the long processes found in the external sulcus, although when seen over their entire length they seem to be associated with capillaries. There is no function other than secretory, as proposed by Shambaugh, suggested for the epithelial cells and the accompanying tubule that fills these periodic gaps in the external sulcus.

In mapping out the intricate vascular system of the spiral ligament Smith[17] describes an extensive network in the spiral prominence. Large arterial vessels enter this region, and the smaller vessels form a narrow band spiraling around with the prominence. We have here described the manner in which the vessels weave in and out among the "root-cells." Smith has pointed out that the network in the stria vscularis, the spiral prominence and upper and lower portion of the spiral ligament are separated by vascular supply and drainage. Small variations in circulation are possible so that disturbances may be confined to one area without necessarily spreading to another. Seymour[18] and Irwin, Weille and Burrage[19] have described such regional disturbances in response to drugs. This possibility, coupled with the findings reported here, points to a mechanism for the control of nutrition to the organ of Corti.

Obviously we do not yet know all about the structures of this region, but by further use of these special staining procedures perhaps more details can be revealed.

Much credit is due Miss Maxine Clapper for her tireless technical assistance in the processing of the material presented here.

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4506 Kresge Medical Research Building.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Arrangements have been completed for the joint meeting of the North Carolina Society of Eye, Ear, Nose, and Throat, and the South Carolina Society of Ophthalmology and Otolaryngology September 17, 18, 19, 1956 Headquarters will be the George Vanderbilt Hotel, Asheville, North Carolina

An unusually attractive program has been arranged, and a large attendance is anticipated.

Asheville, North Carolina, is in the mountains of Western North Carolina, and is a particularly beautiful spot in this season of the year.

For further information write Roderick Macdonald, M D Sec and Treas, 330 East Main Street, Rock Hill, S C

FACTORS AFFECTING HEALING FOLLOWING TEMPORAL BONE SURGERY.*†

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New York, N. Y.

I. INTRODUCTION.

Following surgery of the temporal bone the tendency is for healing to take place in keeping with the natural proclivity of all wounds to heal; nevertheless, even under the best of conditions, the process is slower here than in other regions of the body because of the very nature and location of the postoperative mastoid wound. This comparative slowness of healing, so long as it remains consistent in its progress, does not, however, constitute a problem. In the majority of cases, under competent care, healing is not unduly prolonged. The problem, in postoperative care, arises in the minority group of cases in which there is marked delay or complete arrest in the progress of healing. In such cavities, discharge may persist for many months or even years. This discharge is due to the influence on healing of certain fundamental complicating factors.

It is the purpose of this paper to discuss these adverse factors which have not been clearly understood. In this discussion emphasis will be placed primarily on analysis of the pathophysiology responsible for the difficulties encountered in the postoperative course. Related clinical, surgical, bacteriological, and therapeutic aspects of these problems will also be considered. It is hoped that definition and clarification of the complications which can occur in a post-operative mastoid cavity will help the surgeon in his responsibility for further reducing the number of cases which fail to heal in a reasonable period of time.

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† From the Lempert Institute of Otology, and Lempert Research Foundation, Inc.

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In attempting to explain the poor quality of healing which is sometimes encountered after temporal bone surgery, references have been made to such widely varying influences as the following:

A. *Local Factors.* In otology the surgeon deals with a wound which is a gross operative defect in bone. Unlike the general surgeon, concerned mostly with soft tissue wounds, he does not have the crucial advantage of being able to close his wounds primarily; furthermore, the blood supply, supporting healing processes in a bony cavity, is poor, and being exposed externally, the wound is subjected to lower temperatures which also delay healing. Undoubtedly, these local factors account for the fact that mastoid wounds heal more slowly; but they do not explain why, after the same operation, some ears heal in two or three months while others can continue to discharge indefinitely.

B. *Systemic Factors.* It has been shown that systemic factors have some effect on the progress of healing. Among these general factors are: age, disturbed water balance, abnormal metabolism, avitaminosis, protein deficiency, and anemia.[1] It is true that these factors theoretically and experimentally may be capable of delaying healing. It would probably be missing the problem entirely, though, to accept them as the basis for our difficulties in promoting dry ears. It is understandable how these systemic factors can play a part in delaying healing in individuals with debilitating diseases, severe burns, or states of long-standing malnutrition. It is difficult to believe, however, that such deficiencies can be present in healthy individuals undergoing elective surgery—individuals who reveal no abnormalities on routine preoperative physical and laboratory examinations; furthermore, observations of patients with chronic draining cavities reveal no difficulties in postoperative healing after undergoing surgery in other regions of the body.

C. *Technique of the Surgeon.* Until recent years, practically all of the surgery done on the temporal bone was for infection. When, after a radical mastoidectomy, for instance, a cavity continued to drain for a long period of time, it was assumed that incomplete removal of pathology was responsible for the persistent discharge. This attitude led to many articles in th literature which advocated better surgery as the answer to the problem of obtaining dry, healed cavities. There can be no argument with this basic principle, for certainly if the surgery performed is not extensive or adequate enough to remove foci of infection within the temporal bone, it is unreasonable to expect a permanently dry ear. Today, however, a great many sterile temporal bones are operated upon in the interest of performing functional surgery. In these bones there is no infection to remove and consequently none to remain. Yet we are finding the same resistance to healing in some of these cavities that we have long seen following radical mastoid surgery. Today, also, with the availability of good lighting, magnification, and especially better teaching of surgical anatomy, there are many men who have perfected themselves in the technique of exenterating pathology anywhere in the temporal bone; yet they are discovering that surgical ability, as important as it is, does not necessarily assure dry postoperative cavities. It is becoming more and more evident that the advantage of good temporal bone surgery can be easily lost during the postoperative period where there are fundamental factors which can influence either prompt or delayed healing.

Before discussing these complications of healing it is in order that we first know what constitutes normal healing in a mastoid wound. We should have a working conception of those events of repair which take place in an uncomplicated cavity—one in which healing is occurring under the best possible conditions at the fastest possible rate.

II. THE UNCOMPLICATED CAVITY.

A mastoid wound heals by secondary repair. A substrata of granulation tissue must first develop over the osseous walls before epithelium can spread across the wound to cover the defect.

During the first three or four days following an operation there is an inflammatory reaction in the wound. This reaction elicits exudation of blood elements into the area, resulting in the formation of a coagulum (see Fig. 1) which covers and protects the wound surface. This coagulum, made up of fibrin and other elements of the blood, appears as a reddish, jelly-

Fig. 2. Medium power (270×). Beginning formation of granulation tissue showing new blood vessels and a connective tissue network made up by the fusion of the thin branching processes of the fibroblasts. Clear intercellular spaces contain non staining ground substance. (Histological section of mastoid cavity in monkey twelve days after operation.)

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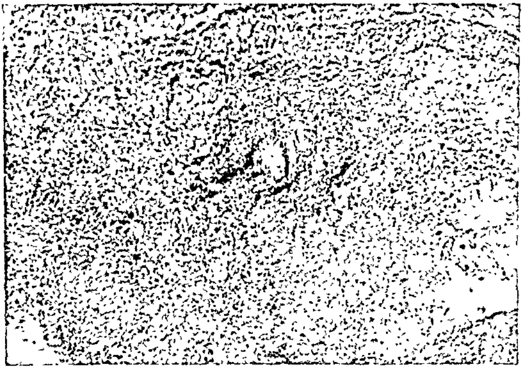

Fig. 1. Medium power (270X). Biopsy specimen of coagulum removed from fenestration cavity on tenth postoperative day.

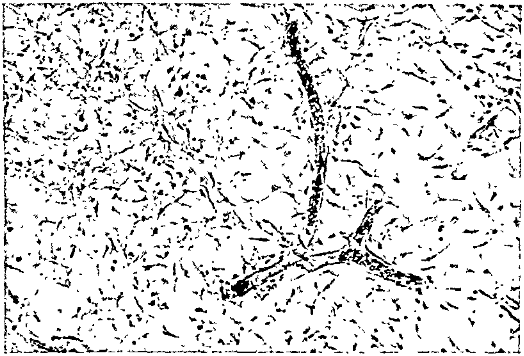

Fig. 2. Medium power (270X). Beginning formation of granulation tissue showing new blood vessels and a connective tissue network made up by the fusion of the thin branching processes of the fibroblasts. Clear intercellular spaces contain non-staining ground substance. (Histological section of mastoid cavity in monkey, twelve days after operation.)

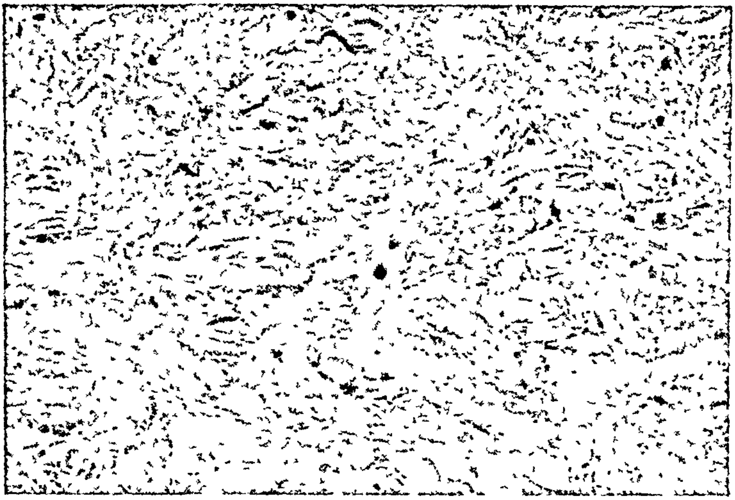

Fig 3 High power (900X) Biopsy specimen of granulation tissue from fenestration cavity on 16th postoperative day, showing elaboration of connective tissue fibrils and elastic fibers Note presence of macrophages and other cells forming a protective and scavenging system

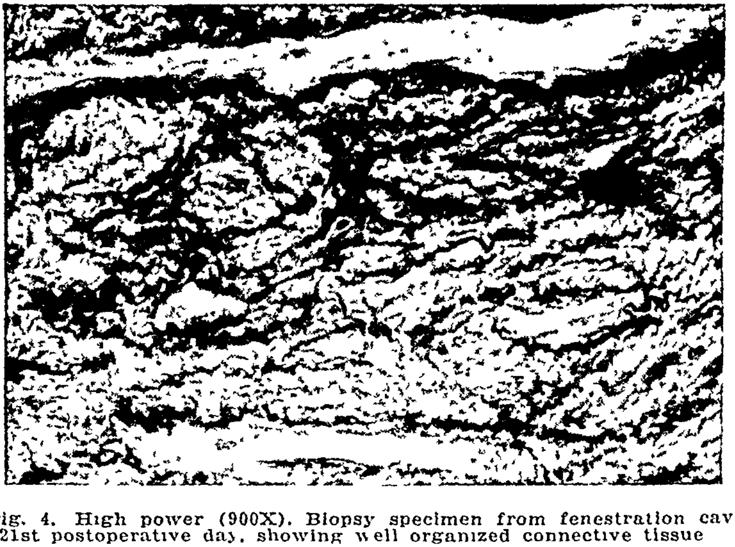

Fig. 4. High power (900X). Biopsy specimen from fenestration cavity on 21st postoperative day, showing well organized connective tissue

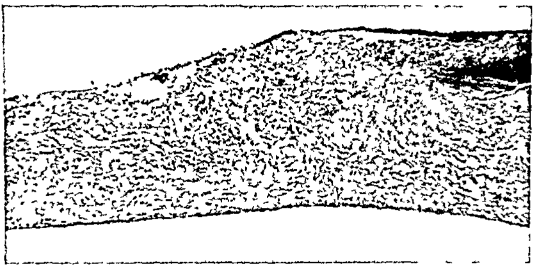

Fig 5 Low power (80X) Biopsy specimen of granulation tissue removed from uninfected postoperative cavity on 30th postoperative day Note smooth healthy surface receptive to the growth of epithelium

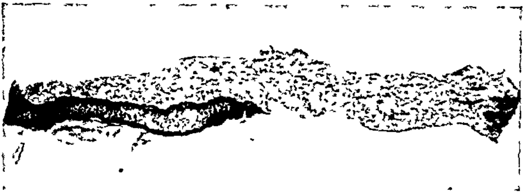

Fig 6 Low power (80X) Biopsy specimen from 90-day postoperative fenestration cavity, showing epithelium growing over uninfected granulation tissue

like exudate. In effect, it provides provisional closure of the wound and offers protection against drying and injury of the underlying structures.[2] Beneath its protective surface the activities of healing begin. These activities are concerned at first, for two or three days, with the removal of injured tissue cells through phagocytosis and enzymatic digestion.[2] Before this period is over regenerative activities have already begun.[3] These are characterized by the appearance of capillaries and fibroblasts which begin to form a sheet of young granulation tissue (see Fig. 2). The capillaries arise from endothelium of surviving capillaries[4, 5] and the fibroblasts are either brought in with the blood elements or migrate from surviving connective tissue in the area.[6] The young granulation tissue, as it develops, offers a better protection to the injured osseous walls than the jelly-like exudate[7] which now gradually disappears as its fibrinogen content decreases, and a resorption of the exudates takes place. By the end of the third or fourth postoperative week most of the exudates have been removed, and a thin sheet of granulation tissue, covering the walls of the cavity, has become well organized (see Fig. 4). It now consists of new capillaries and a connective tissue network, made up by the fusion of the thin branching processes of the fibroblasts (see Fig. 2). The intercellular spaces of the network contain an amorphous jelly known as ground substance (see Fig. 2). Angevine[8] has likened the situation to "absorbent cotton with the ground substance representing the interstices between the cotton fibers." This extremely interesting substance is present in varying amounts depending upon whether the tissue is young or old, suffers injury, or becomes infected. The fibroblasts are responsible for the elaboration of the connective tissue fibrils[9] and the elastic fibers[10] which become embedded in the ground substance (see Fig. 3). As the connective tissue matures the fine fibrils coalesce to form larger collagen fibers which, in turn, form sheets of collagen. The direction of the collagen fibers is determined by the lines of tension in the tissues.[11,12] In addition to these main elements of the connective tissue structure, other cells such as macrophages, mast cells, lymphoid cells, plasma cells, and eosinophils may be seen[13] (see Fig. 3). Tissue fluids, derived from the plasma, are also present.[14]

As long as the connective tissue remains uninjured and uninfected it offers a smooth, firm, receptive surface for the implantation of new epithelium (see Fig. 5). Epithelium begins to grow over the newly formed granulation tissue (see Fig. 6) by a process of cell division and migration from the wound edges and from any skin which may be present in the bowl of the cavity. Healing is accomplished only when a continuous layer of epithelium has covered the new granulation tissue. Following closure of the wound, cell division may continue after migration has ceased, adding thickness to the covering epithelium.[15]

Management of the Uncomplicated Cavity. Now, how should a cavity that is healing without complications be treated?

First of all, it should be realized that there is absolutely no evidence to support the multitude of claims that various substances applied to wounds stimulate healing.[1] Although wounds have engaged the attention of physicians for centuries, not one single method has been discovered for speeding up nature's timetable of healing beyond the optimal rate under ideal conditions.[16] As long as a cavity is healing without complications there is nothing a surgeon can do to improve on the biological processes taking place. The great temptation to treat a cavity, on the other hand, may result in a number of factors which interfere with the natural tendency for wounds to repair themselves. These effects, which may be brought about by manipulation in the cavity, are:

Removal of the Elements Needed for Healing. Cleaning a cavity, in the early stages of healing, only removes the exudates and the blood elements present, which nature put there in the first place, to accomplish the events of healing.

Trauma. After healthy granulation tissue has formed, wiping the cavity with a cotton-tipped applicator, except in the gentlest manner, causes traumatic inflammation and edema of the newly formed granulation tissue.

Infection. Unless manipulations in the cavity are carried out under strict aseptic techniques they may be responsible for innoculating the uncovered granulation tissue with bacteria. In some cases, insufflation of an unsterile powder may introduce more infection than it prevents.

Destruction of Tissue Cells. Some antiseptic agents applied to a wound may have destructive action on living tissue as well as on bacteria.[17]

Damage to New Epithelium. Soon after a cavity has become healed, new epithelium, which is at first more firmly adherent to the overlying scab than to the underlying connective tissue,[18] may be pulled away when the surgeon is too eager to remove crusts.

An uncomplicated cavity should be thought of in terms of biological and histological events and not in terms of mechanical cleanliness. It is becoming more and more obvious that a good attitude to assume toward uncomplicated healing is one which might be called "judicious neglect."

III. THE COMPLICATED CAVITY.

There are four fundamental complicating factors which can delay permanent healing in a postoperative mastoid cavity. They are:

A. Bacterial infection.

B. Mycotic infection.

C. Cyst formation.

D. Cicatrization.

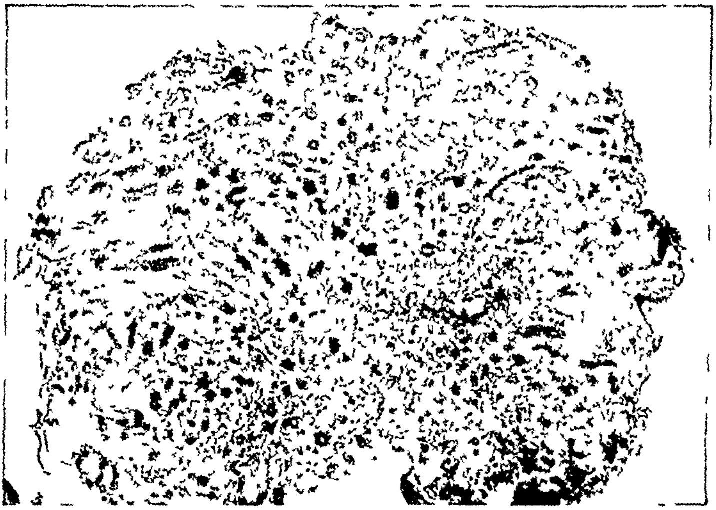

Fig 7 Very low power (25X) Typical "humped up" infected granulation removed by curettage from a purulent cavity Note marked accumulation of intercellular fluid, increased vascularity, and heavy infiltration of leucocytes

I choose to call these fundamental factors because in all unhealed ears with long continued drainage, one or more of these four factors will be found responsible for the discharge. These are the patho-physiological complications which explain the tremendous variation in the healing properties of different cavities. They are factors which have great clinical importance, and over which the otologist has some measure of control.

A. *Bacterial Infection.*

By far the most frequent complication in the healing mastoid is bacterial infection. During the postoperative course many chances for contamination occur in a wound which requires two or three months for complete epithelization. It is not economically practical to keep a patient hospitalized until

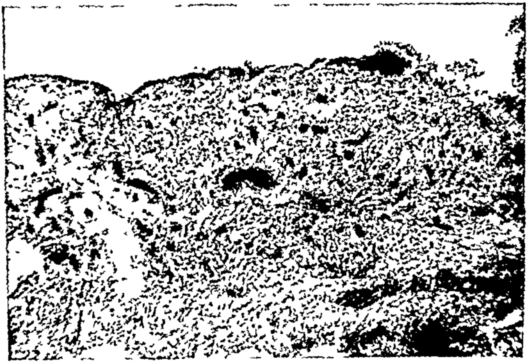

Fig 8 Low power (80X). Biopsy specimen from cavity with infection for a period of ten months Note increased vascularity, accumulation of intercellular fluid, and heavy infiltration of leucocytes in the superficial layer of infected granulation tissue Collagen is building up in the deeper layer of connective tissue

healing is complete, and patients refuse to wear a sterile bandage for long periods of time, calling attention to an operation involving a sense organ; furthermore, long term prophylaxis with antibiotics is neither wise nor safe.

Once infection has become established in
elaborated by the invading organisms.
damage to the healthy granulation tissue.[1]
injured granulation tissue undergoes a co
tive response, to prevent spread of the
areas. This response is characterized by se

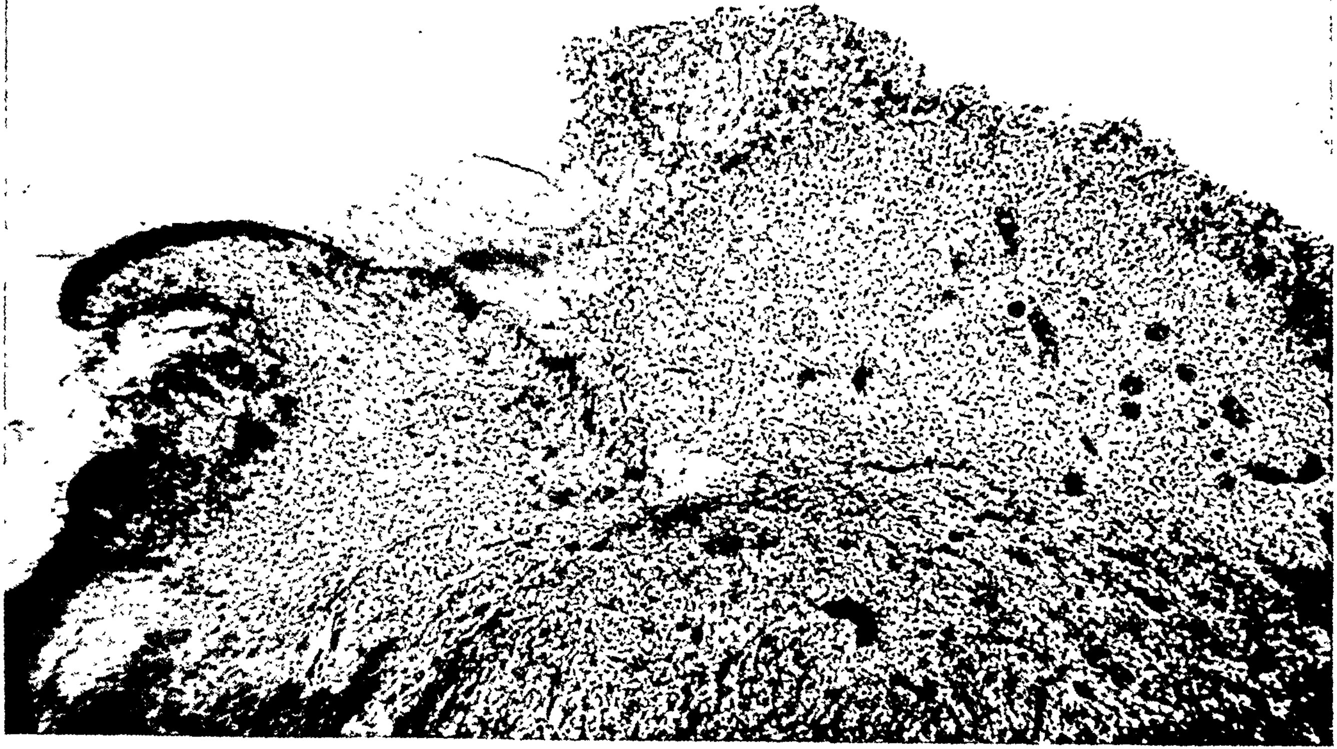

Fig. 9. Low power (80X). Biopsy specimen from infected postoperative cavity showing arrest of healing. Epithelial growth (left), which has been extending over a healthy bed of connective tissue (bottom) ceases to grow further on meeting an area of infected granulation tissue (upper right). Epithelial cells pile up at the margins of the wound forming a thick border and further impeding growth of cells across the wound surface. Note exudate on surface of infected granulation tissue.

Increase in the Amount of Ground Substance (see Figs. 7, 8). This intercellular substance in general acts as a barrier against the spread of infection, and an increase in the amount present acts as a further protection.

Increased Vascularity (see Figs, 7, 8).

Influx of Leucocytes and Phagocytes (see Figs. 7, 8).

Active Exudation (see Fig. 9).

All of these activities are ideal responses for a defense against infection.[19] In principle, then, the edematous granulation tissue represents a barrier against bacterial invasion and toxin absorption.[7,17] Its protective presence accounts for the clinical fact that a postoperative cavity can drain pus for months or years without the patient's showing any systemic effects;[20] however, epithelium will not grow over such infected granulation tissue. Unless epithelial cells have healthy granulation tissue on which to implant themselves, the cells pile up at the margins of the wound, forming a thick border which further impedes the growth of cells across the wound surface[21] (see Fig. 9). As long as infection remains in the cavity growth of epithelium does not take place, and the progress of healing is arrested.

Infection should be prevented, of course, in so far as possible. It seems unnecessary to go into those details concerning the need for sterile precautions in the care of an open wound during postoperative dressings and office visits. Every surgeon should be aware that this is an important part of his responsibility. It is only realistic to understand, however, that in the activities of the patient there are many circumstances leading to infection which are beyond the surgeon's control. Anyone who does much temporal bone surgery must expect to contend with the problem of infected cavities.

Necessity for Removal of Infection to Reestablish the Progress of Healing.

After infection has occurred there is no longer any justification for maintaining an attitude of "judicious neglect". Active measures must be taken to eliminate this complication if the progress of healing is to be reestablished. The cavity must be cleaned and the areas of pathology determined.

Mechanical measures, such as the common practice of curetting away the edematous granulation tissue, accomplishes little unless at the same time infection is eliminated. Without the elimination of infection, the toxins of bacteria still present, again injure the connective tissue on the surface of the curetted base, and within a short time granulations reappear in the same area. This cycle, with curettement and reappearance of infected granulation tissue, can go on indefinitely as long as suppuration remains in the cavity.

The Problem of Eliminating Postoperative Mastoid Infection.

This problem is related to: *1.* the types of bacteria present, *2.* the resistance of these bacteria to therapy, and *3.* the difficulties of delivering sufficient concentrations of a drug to the site of infection.

1. *Types of Bacteria Present in Postoperative Mastoid Cavities.*

In order to satisfy myself as to the general distribution of bacteria found in infected postoperative mastoid cavities, I collected and studied a series of cultures from consecutive patients being seen in the course of routine postoperative office care. These were cultured aerobically and anerobically. Dr. Philip Meltzer[22] has made a similar study at the Massachusetts Eye and Ear Infirmary. In summary, both these studies showed that approximately 50 to 60 per cent of organisms cultured were staphylococcus aureus, coagulase positive. The large majority of the remaining organisms were of the pseudomonas or proteus types.

These organisms, with others such as fungi and E. coli, are bacteria which seem to be present fairly consistently in the normal flora of the skin.[23,24,25] Finding them in mastoid wounds simply reflects the opportunities for contamination from the skin of the patient or the hands of medical personnel treating the ear.

2. *Resistance of These Bacteria to Therapy.*

A further explanation for their presence, consistently, in mastoid wounds is that these are the bacteria which develop as superimposed infections in wounds in general, as a result of antibiotic suppression of more drug-sensitive microorganisms. As Cannon[26] has pointed out, one of the complications of greatest current interest is the increasing frequency with which we are being confronted with infections attributable to staphylococci, pseudomonas aeruginosa, and proteus, all resistant to most of the antibiotics now in use.

3. *Difficulties of Delivering Sufficient Concentrations of Therapeutic Agents to the Site of Infection.*

It is generally recognized that there are few instances of infection in the body which cannot be more effectively reached by way of the blood stream than by locally applied agents.[27] When a mastoid wound grows older, however, and has been the site of infection for a long period of time, it develops characteristics which limit the effectiveness of systemically administered agents. Where healing has been slow, scar tissue formation takes place in the deeper layers of connective tissue (see Fig. 8). This deeper connective tissue becomes thickened, sclerotic, and poorly vascularized, and blood-borne drugs reach the surface layer of infected granulation tissue in poor concentrations.[28] MacLeod[29] has stated that such an area of infection becomes "essentially walled-off from the general circulation." I have seen this confirmed clinically many times, as evidenced by the inability of parenterally administered antibiotics to control infection in old postoperative mastoid cavities although present in inhibitory concentrations in the blood.

Rationale of Therapy. There is a solution to this problem of overcoming drug-resistant types in an area of infection surrounded by a fibrous barrier. This calls for a design of treatment which will deliver a wide-spectrum antibiotic to the site of infection in such form that it will remain there a sufficient length of time to accomplish maximal exposure of the organism to the drug. As Davis and McDermott[30] have pointed out, susceptibility of an organism to a chemotherapeutic agent is a quantitative rather than a qualitative attribute; therefore, if a chemotherapeutic agent is rendered at a high enough concentration, it can inhibit practically all strains of bacteria. In other words, if we can deliver a sufficently high concentration of an antibiotic at the site of infection, we can get not only wide antibacterial activity but also activity even against organisms which are not usually susceptible to lesser concentrations of the same drug, and we can overcome drug resistant types.

Delivering high concentrations of a blood-borne drug to the site of a chronic infection in the postoperative mastoid cavity is difficult, however, not only because of the presence of the fibrous barrier surrounding the site of infection, but also because of the danger of potential systemic toxicity; therefore, local therapy becomes the route of choice in old suppurative mastoid cavities.

In general, however, the common fault of local therapy is that it is carried out in concentrations which are too weak. As Bunn[31] has stated, success in the management of any infection depends upon exposing the organism to sufficient concentrations of its antagonist. Where the infection is due to drug-resistant bacteria, higher concentrations of a drug must be used. A criticism of most of the commercially available agents for local therapy is that they are supplied in strengths that will meet the demands of a wide market. For instance, otic drops are supplied in strengths of one-half to one per cent; ointments in strengths of 3-5 mg./Gm. Obtaining sufficient concentrations of an antibiotic at a particular site of infection for any length of time is, therefore, difficult. Effective concentrations, sufficient to end an infection rapidly even in the presence of drug-resistant organisms, can be produced best by introducing an antibiotic in the form of a concentrated powder, delivering to the site of the infection as much as 250-500 mg. of a drug.

Management of the Cavity Complicated by Bacterial Infection. The approach to sterilizing suppurative cavities is illustrated in the following clinical experience, which is presented, not as justification for choosing a specific agent, but rather as an explanation of the preceding rationale of therapy.

About five years ago, after running sensitivity tests on a series of cultures collected consecutively from infected postoperative fenestrat[illegible] routine office practice, I selected chloramphenicol [illegible] clinical trial because of its broad [illegible] and its effectiveness against proteus [illegible], and resistant staphylococci. Since then I have [illegible] 657 cases of postoperative suppurative fenestration [illegible] radical mastoid cavities in which the infection was not previously controlled by less effective routine measures. Treatment is carried out in the following manner:

> "Infected granulations, which are "humped up" and interfere with the contour of the cavity, are removed by curettage and the denuded area dried with cotton packs moistened with adrenalin. The powder from a 250 mg. capsule of chloramphenicol is then dropped into an ear speculum and worked into the cavity to the unepithelized area or areas with a cotton-tipped probe. Care is taken to see that all unepithelized areas are thickly covered with the powder, since infection in the entire cavity must be eliminated at one time to prevent foci of infection from remaining. For instance, patients with denuded areas on the tegmen must be placed in such position for treatment that the powder may be transferred from the speculum to this area. Two capsules (500 mg.) may be used if the denuded area is extensive. The powder mixes with secretions and soon becomes a caked paste which, in two or three days, becomes a dried crust. Epithelization of the sterilized area takes place beneath the crust. One or two treatments, one month apart, are usually sufficient to render a cavity aseptic. It is unnecessary to see the patient frequently. As a matter of fact, once the cavity is dry, the crust should not be disturbed for a month or six weeks, since lifting up the crust will pull away the new epithelial tongues which are at first more firmly adherent to the crust than to the underlying connective tissue.[18] Success with this method of treatment has been surprisingly consistent, not only in infections with the Gram-negative group, but also in wounds infected with Gram-positive organisms."

In 657 cases of persistent discharge treated, 538 (82 per cent) cavities became dry and completely epidermized after the following number of treatments:

No. of Treatments	No. of Cases	Percentages
1	310	57
2	154	29
3	53	10
4	16	3
5	3	.5
6	2	.5
	538	100.0

In the remaining 119 cases (18 per cent) the treatment was not carried to successful completion because of: *1.* development of reactions; *2.* cessation of treatment due to the fear of reactions; *3.* association of the infection with other complications in the same cavity (cysts, fungus); *4.* cases which could not be followed, and *5.* failures.

Sensitivity of the patient to chloramphenicol developed in 36 (5.5 per cent) of the 657 cases treated. This reaction to the use of chloramphenicol in heavy concentrations was no greater, in my experience, than that encountered when weak solutions of drops were used. Sensitivity developed on a quantitative basis, 70 per cent occurring in cases where the drug was used more than three times. This small percentage of reactions can be further reduced if the clinician will realize that he has a remarkable instrument for rendering a cavity aseptic but will not prolong treatment if the infection does not readily respond. (There is very little to be gained by prolonging the treatment in any event. It is significant that 517 (96 per cent) of the 538 cases successfully treated required no more than three treatments.)

The possibility of sensitization with the use of chloramphenicol cautions against the indiscriminate use of this antibiotic. This disadvantage prevents its becoming what might be an ideal agent for our purposes. We do not yet have an ideal antibiotic for local use, one which could be easily introduced into a cavity, in such form that it would remain active in the infected area for a long period of time, in sufficient strength to be effective over a wide range of bacteria, without sensitizing the tissues.*

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* Reports on newer antibiotics appearing all the time make any estimate of this situation unpredictable. A recent report[32] cites the use of erythromycin and oxytetracycline in pyrogenic infections of the skin without any evidence of sensitivity.

Other forms of antibacterial treatment, which do not involve the small calculated risk of sensitivity, are preferable in early postoperative cases or may be necessary in instances where sensitivity has already been encountered in old chronic draining cavities. When the identity of the infecting organism is known to be staphylococcus, large concentrations of bacitracin powder may be tried. Sensitivity to this agent, when used locally, is practically unknown. Aerosporin may be used in the same way, without risk of a sensitizing effect, when the infecting organism is known to be pseudomonas aeruginosa. Other treatments, eliminating the possibility of sensitizing effects, consist of the use of various ointment-combinations of bacitracin, polymyxin, and neomycin. In general, these preparations, in standard forms, are less effective against postoperative mastoid infections because of their weak concentrations. Results, however, are enhanced when quarter-inch packing is impregnated with these ointments, and the cavity is firmly packed. Twyman[33] 33 years ago showed that epithelium will continue to grow when subjected to the same pressure which collapses the capillaries in granulation tissue and thereby suppresses its growth.

B. *Mycotic Infection.*

Another complicating factor which can delay healing in a postoperative mastoid cavity is mycotic infection which flourishes in a bacteria-free field, and is more prone to appear in cavities after effective antibacterial treatment has been carried out. Aspergillus, as well as some other molds (Penicillium and Mucor species) are responsible for stubborn, chronic infections in postoperative wounds. Practically, however, the problem may be narrowed to Aspergillus niger in particular. In my experience, this has been by far the most frequent type of mycotic infection encountered in postoperative cavities. When this contaminant occurs it is obvious that it must be differentiated from a bacterial infection, since no amount of antibacterial therapy will affect the situation. Clinically it is easily recognized by the presence of a material resembling moist gray blotting paper, on the surface of which may be seen the black spore heads so characteristic of this type of fungus. The patient complains at first of intense itching, followed by discharge and pain. When cleaned the cavity has a degree of inflammatory reaction paralleling the duration of the infection.

Management of the Cavity Complicated by Mycotic Infection.

In my own experience, treatment of fungus infections in postoperative cavities, once very difficult, has ceased to be a problem since I began using an iodine compound, Vioform cream (3 per cent). This appears to be an ideal agent in these infections. I have now used it in 93 cases, without failure to eliminate fungus infection readily and easily, and without discomfort to the patient.

It is used in the following manner: All debris and membranes are first removed by mechanical cleansing. The cream is introduced into the cavity with a syringe to which is attached a piece of flexible plastic tubing. It is then worked into all recessed areas with a cotton-tipped applicator. This is followed by filling the entire cavity (leaving the fenestra clear, in fenestration cavities). One treatment usually soothes the inflammatory reaction of the tissues and eliminates the fungus. An additional one or two treatments are given one week apart to insure against return of the infection.

C. *Cyst Formation.*

A third complication that can occur to prevent permanent healing of a postoperative cavity is cyst formation (see Fig. 10). This results in a type of discharge which, unlike the preceding complications, is not caused by an invading organism, but is caused by a secreting epithelium, either squamous, cuboidal, or columnar.* Consequently, recognition of this type of drainage becomes essential to insure proper treatment. The discharge has a characteristic mucoid appearance, and is tenacious and stringy. As one removes it from the ear it has the appearance and consistency of egg albumin. Some cysts will be found filled with a chocolate-colored hemorrhagic fluid. These cysts form most frequently in the sino-dural angle or in the mastoid tip. Skin over the cyst appears bluish, indicating a mucus-filled space beneath. They may appear months or even years after operation, more often after the cavity has been dry for a long time. The clinical course is usually one of alternating periods of mucoid discharge with periods when the ear is dry. When a cyst is small, and located in such a position in the cavity that it cannot be visualized, the nature of the discharge may not be readily determined, especially if there is superimposed suppuration.

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* I have biopsied one cyst from a mastoid cavity which contained no epithelial lining at all, the cavity being surrounded by fibrous tissue. This finding may be explained by the effect of increasing pressure of the cyst contents on the epithelium until it becomes atrophic and disappears entirely.[34] The presence or absence of an epithelial lining in a cyst also explains why some cysts continue to enlarge and rupture, while others do not.

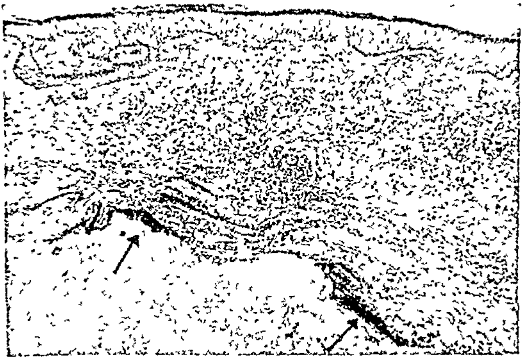

Fig 10 Low power (55X). Biopsy specimen from fenestration cavity containing a cyst seven years after operation The cyst cavity, filled with cystic material, is at lower left Arrows indicate cystic lining

The etiology of these cysts has been puzzling. One explanation is that they are retention cysts, formed during the process of healing when epithelium covers over a mastoid cell containing mucous membrane. If this were the etiology, it seems unlikely that any cavity could heal without subepithelial cyst formation, since there are always some cells left somewhere in a postoperative cavity. A more likely explanation is that cysts are the result of atypical epithelial growths. Arey[35] believes that cyst formation may occur in granulating wounds which have been injured. In other words, in a cavity which has been the site of infection or much trauma, the connective tissue may become subnormal. Epithelial processes may extend downward into this injured connective tissue, forming an atypical growth, or as Arey has stated,[35] "can invade growing connective tissue such as is active in filling a defect."

Whatever the etiology, the fact that active, secreting, subepithelial cysts can occur in postoperative cavities is a reality. Such cysts will usually rupture when they become distended, furnishing the source of a persistent or intermittent discharge; at other times they may fail to rupture, causing pressure dissection instead. This can result in lifting up a considerable area of skin from the osseous wall causing pain, symptoms of pressure, and in the fenestration cavity, dizziness.

Management of the Cavity Complicated by Cyst Formation.

In the observation of approximately 2000 postoperative fenestration and radical mastoid cavities during the past five years I have encountered 52 cysts requiring treatment (approximately 2.5 per cent).

No amount of antibacterial treatment has any effect on cystic drainage. The cystic membrane must be eliminated. Although surgical intervention in the cavity makes complete removal easier, the patient can usually be spared an operative procedure. In the majority of cases, destruction of the cystic epithelium may be accomplished as an office procedure in the following manner: As much as possible of the presenting face of the cyst wall is removed with cutting or biting instruments. The remainder of the cyst cavity is then packed, with narrow iodoform gauze weekly, for two or three weeks. This keeps the lumen of the cyst open, converts it to a part of the cavity lining, and destroys the secreting epithelium. A dry cavity then becomes possible unless there is a superimposed purulent discharge present, in which case antibacterial therapy must also be carried out to obtain epithelization of the destroyed cystic area.

D. *Cicatrization.*

When a mastoid cavity has been the site of long delayed healing, a fourth factor comes into play, which makes it more difficult to obtain *permanent* healing. This factor is excessive cicatrization. Cicatrization is essentially the conversion of granulation tissue into scar tissue. When chronic infection persists for a long period of time there is repeated damage to the granulation tissue by toxins of bacterial organisms present. This leads to repeated episodes of necrosis in the tissues. More and more fibrous tissue reaction follows, and in the course of time an increasing amount of scar tissue forms. This is the most characteristic feature of chronic inflammation.[36] As the scar tissue undergoes shrinkage, capillaries regress, and vascularity decreases. Such scar tissue supports a poor quality of epithelium. It is thin, flat, and atrophic (see Fig. 11), and is easily wiped away because it does not have rete pegs attaching it firmly to the connective tissue.[37]

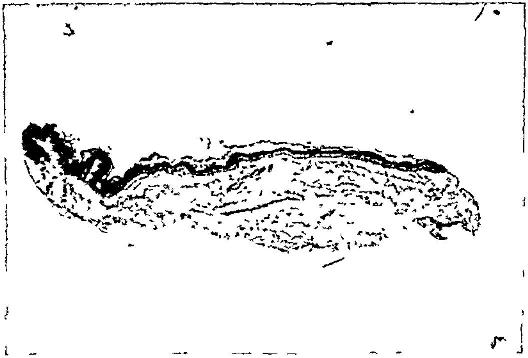

Fig. 11. Low power (80X). Biopsy specimen from fenestration cavity seven years after operation showing poor quality of epithelium over sclerotic connective tissue. Note absence of rete pegs and thin, flat atrophic nature of epithelium.

Management of the Cavity With Excessive Cicatricial Healing. In the cavity where healing has been prolonged one should be aware of this histological situation. Extreme gentleness is necessary in removing crusts from the dry ear, or in periodically removing accumulated wax, in order to prevent traumatiz wipe away the epithelium, leaving a denuded area which may become infected and reestablish a discharge.

IV. REMARKS ON SURGICAL TECHNIQUE.

A. *Need for an Adequate Opening to the Cavity.*

An adequate opening is obviously essential if the otologist is to exert any measure of control over complications which may take place during the events of healing. Failure to provide for it at operation is often responsible for complete defeat in efforts to obtain a healed, dry cavity. Nothing is so frustrating or so difficult as trying to carry out instrumentation, or trying to apply medication to a cavity blindly, through a small constricted opening. The principal factor in atresia of endaural openings is a prominent inferior spine (the spine existing between the external auditory canal and the exenterated mastoid after the posterior bony canal wall has been removed). The upper circumference of endaural openings, no matter how extensive the incisions, will heal more or less at the same level because of the conformity of the cartilagenous components of the auricle to fixed positions. Consequently room for an adequate opening must be provided by removing bone inferiorly, which should include not only the inferior spine but also a lowering of the floor of the external auditory canal. Adequate diameter of the opening in an antero-posterior direction may be assured by removing a small semilunar piece of cartilage from the anterior edge of the conchal cartilage. With the routine postoperative use of antibiotics the threat of perichondritis with this procedure is minimal.

B. *Size of the Cavity.*

Emphasis on the reasoning that a limited cavity* assures more rapid healing because there is less surface area to be epithelized, has been out of proportion to its importance. We know, on the one hand, that regenerating epithelium extends faster in smooth and shallow wounds than in uneven wounds, or where there are mechanical obstacles to the movement of cells.[38] For this reason it follows logically that the limited cavity with smooth surfaces favors healing. This favorable factor for the spread of epithelium pertains, however, only as long as the healing remains uncomplicated. The introduction of infection affecting the surface layer of granulation tissue, for instance, can delay and arrest healing in a small or a large cavity; and it is one of the fundamental factors such as this which is usually responsible for long-delayed healing—not the difference in the amount of surface area to be covered by epithelium.

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* The term *limited cavity* is used here in the sense that cells posterior to the sinus plate and cells below the level of the floor of the external auditory canal are not removed.

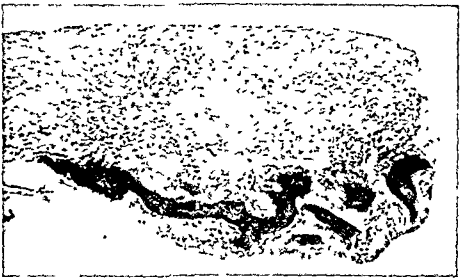

Fig 12 Low power (80X) Biopsy specimen from fenestration cavity showing how small cellular defects in the osseous wall of the cavity are filled in with granulation tissue

What is of more importance is that a limited cavity is easier to take care of postoperatively because all surfaces are more easily visualized. In the event that complications develop they can be observed and properly treated.* A large cavity, on the other hand, may even be difficult to take care of routinely. When complications appear, one may be defeated in his efforts to obtain a dry ear because he is not only unable to visualize the pathology but is also unable to apply medication to recessed areas, unless a very large endaural opening has been provided for at operation.

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* It has been suggested that retained cells in a cavity following fenestration may become the site of a postoperative mastoiditis Like Day[30] I have had no such experience, nor have I ever found it necessary to revise a cavity because of infected cells in order to get a dry ear Granulation tissue fills in small defects (see Fig 12) and the overlying bed of granulation tissue acts as a barrier to infection to protect underlying osseous structures

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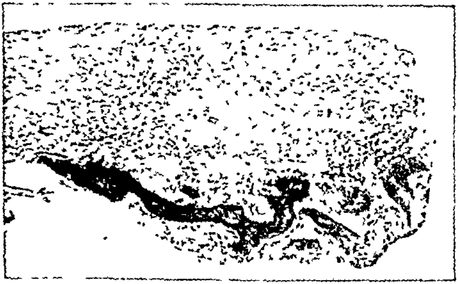

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There is another side to the question. It is not always wise and it is not always easy to limit the size of a cavity. In doing a radical mastoidectomy, where there is chronic mastoiditis, the amount of bone removed must be governed by the extent of the infection. In the performance of the fenestration procedure one encounters all types of cellular development. In a large pneumatic or pneumosclerotic bone it is sometimes very difficult to limit the size of the cavity. without leaving large open cells. The resulting irregularities of the cavity wall, too large to be filled in completely with granulation tissue, may predispose to cyst formation by causing atypical sac-like epithelial growths. When one cannot do a *good incomplete* exenteration without leaving large open cells he should always do a *good complete* exenteration. In such cases it is necessary to exenterate all the cells, including retrosinus and tip cells, before smooth limits of the cavity can be obtained.

C. *The Role of the Skin Graft.*

If healing is attained when a wound surface is completely covered with epithelium then, at least from the theoretical point of view, it would seem that skin grafting should long since have solved all the problems of postoperative care. That it has by no means accomplished this is indicated by the indifference toward its use which still exists today, many years after introduction of this technique for decreasing the difficulties of postoperative care. Among otologists with whom I have discussed this question it is surprising to find how many have given up this procedure after having employed it at one time or another. This attitude is difficult to understand, in view of the number of recent reports in the literature, by some otologists, indicating increasing success and enthusiasm for skin grafting following temporal bone surgery.

It has been pointed out recently by Campbell[40] that the chief cause of failure with skin grafting in mastoid cavities has been the lack of care and time taken in obtaining and placing the grafts. On the other hand, those surgeons who have abandoned skin grafting, after a period of trial, usually explain that a reasonable measure of the success of skin grafting is whether or not a dry cavity is obtained in a shorter period of time and that since the postoperative course is not noticeably different in enough cases to warrant the added trouble of this procedure, they have given it up.

It is not to be doubted that skill and careful attention is necessary in obtaining the grafts and applying them in intimate contact with all the contours and recesses of a cavity. I am far from convinced, however, that the ultimate fate of the grafts rests entirely on the skillful technique of the surgeon, or that ability in this procedure varies so considerably among a number of highly experienced otologists.

I believe that the real fundamental factors, one leading to the other, which are responsible for reducing the practical results of such a theoretically sound procedure as skin grafting are: *1.* the difficulties of getting a "take" of skin grafts to bone, and *2.* the hazards of infection to which the postoperative cavity is exposed at any time between operation and the complete epidermization of the wound surface. The most skillfully obtained and applied skin graft is still subject to the hazards of poor blood supply from bone, unfavorable pressures by packing, serum dissection, shrinkage, and, in radical cavities, sloughs due to infection. The net result of the amount of skin which "takes" in the usual so-called completely grafted cavity is considerably less than 100 per cent. This leaves bare areas of bone in the postoperative cavity which must be healed by the same process necessary in ungrafted cavities—first by the formation of a substratum of granulation tissue, followed by the slow growth of epithelium from adjacent skin margins over the unepidermized area. Thus, as in the ungrafted cavity, for a period of time after operation, there is uncovered granulation tissue which is vulnerable to infection. When infection does occur, delay and arrest of healing can take place to the same extent as in the ungrafted cavity. It must be remembered that a cavity does not become healed and dry until complete epidermization has occurred, and that a small area of infected granulation tissue can keep a cavity draining for as long a period of time as a large area. The problem is the same. Infection must be eliminated before healing takes place. It is true that if infection does not intervene, grafted cavities have the advantage of healing faster than ungrafted cavities. Under the same circumstances, however, a cavity without free grafts heals fast enough not to present a problem.

Usefulness of free grafting in mastoid cavities is probably better illustrated by other more fundamental advantages. Even if the "take" of skin is only partial and infection does occur in the cavity, scattered areas of grafted skin play an important role in preventing the cavity from filling in with fibrous tissue. In an infected cavity where there is no skin, there is a tendency on the part of nature to heal the wound defect by second intention. It has probably been the experience of every otologist, at one time or another following a radical mastoidectomy, to observe a cavity containing no skin fill in with fibrous tissue despite all his efforts to prevent it, and finally to close off at the very surface of the wound margins. This is brought about by the fact that pus is more detrimental to the growth of epithelium than to the growth of connective tissue.[41] The growth of epithelium, arrested by the presence of suppuration in the wound, does not bridge across the cavity until it has filled in with connective tissue giving the epithelium the advantage of a more exteriorized environment where it is less affected by the presence of suppuration.

Another valuable advantage of grafting is that the Eustachian tube orifice may be sealed off in a radical mastoidectomy by grafting over the orifice. This is essential to prevent repeated discharge in the cavity with each upper respiratory infection, the occurrence of which makes it difficult to obtain a permanently dry cavity. A skin grafted cavity also results in less scar tissue beneath the epithelium overlying the areas of successful "take".[42] The advantages of this are discussed in the following section.

The use of local antibiotics in combination with surgical packing, as well as during the postoperative course, has undoubtedly been responsible for reports of increasing success with skin grafting. It should not be overlooked, however, that the local use of antibiotics has also been an enormous advantage in obtaining dry, healed cavities, without free grafts.

V. REMARKS ON THE HEALED CAVITY.

A cavity may be considered as healed when the connective tissue is completely covered by a continuous layer of epithelium. The processes of healing, however, do not stop at this stage, but continue for many months thereafter,[43] consisting of maturing changes in the connective tissue. These maturing changes are brought about by a reduction in the amount of intercellular fluid, and a decrease in the number of cells and blood vessels. The protective reactions of increased intercellular fluid, increased vascularity, leucocytosis, and phagocytosis (see Figs, 7, 8) were needed in the uncovered granulation tissue as a barrier against infection. After epithelization provides protection to the connective tissue, these defensive reactions are no longer needed and gradually disappear. The result is a change of the granulation tissue to scar tissue. Capillaries regress, the intercellular fluid diminishes, and the loosely arranged fibrils and collagen fibers consolidate into thicker and more closely packed collagen bundles. Clinically, after epithelization, we can follow this change by observing the thicker, *pink (but dry) soft tissue of the cavity gradually take on a* thinner pearly-white appearance after a few months.*

These maturing changes, which take place as a natural course of events in all connective tissue contributing to the processes of healing, have little clinical significance when the healing has been uncomplicated, with deposition of connective tissue limited to the amount consistent with repair; however, when there has been a long delay in healing due to chronic infection, repeated damage to the granulation tissue stimulates the formation of more and more granulation tissue which gives rise, under pathological conditions, to collagen[44] in the deeper layers of connective tissue (see Fig. 8).

As Converse[42] has stated, "As the unhealed wound grows older this deep layer of fibrous tissue tends to increase at the expense of the superficial layer of granulations." The clinical effects of this thicker layer of connective tissue, covering the walls of the cavity, remain unnoticeable before complete epithelization has occurred. This is because the protective presence of large amounts of intercellular fluid, in the presence of an inflammatory stimulus, keeps the tissue loose and elastic (see Figs. 13, 14); however, after complete epithelization of lization and then gradually disappear. The syndrome may be abolished at any time if the epithelium breaks down and infection is reestablished in the cavity.

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*In the occasional healed fenestration cavity the fenestra may be seen through the overlying skin almost as easily as looking through cellophane. This is accounted for by the fact that the disappearance of cells, fluid, and blood vessels leaves a more homogeneous tissue (made up almost entirely of a thin layer of collagen). Light striking the tissue is not refracted by several elements of different densities and is more readily transmitted.

Among the extensive observations by Travell[46] on skeletal muscle pain and the trigger mechanism, she has described an almost identical composite pattern of pain, with concomitant autonomic effects, in a series of patients in whom a clinically related trigger area could be demonstrated in the sternomastoid muscle. The pattern of referred pain from the trigger area is essentially the same from person to person. Predisposing conditions leading to establishment of the trigger area were some form of trauma to the muscle, physical, infectious, thermal, or metabolic. Inserting a needle into the trigger areas gives rise to the characteristic pattern of referred pain. Infiltrating the trigger area with procaine abolishes the pain in the reference area.

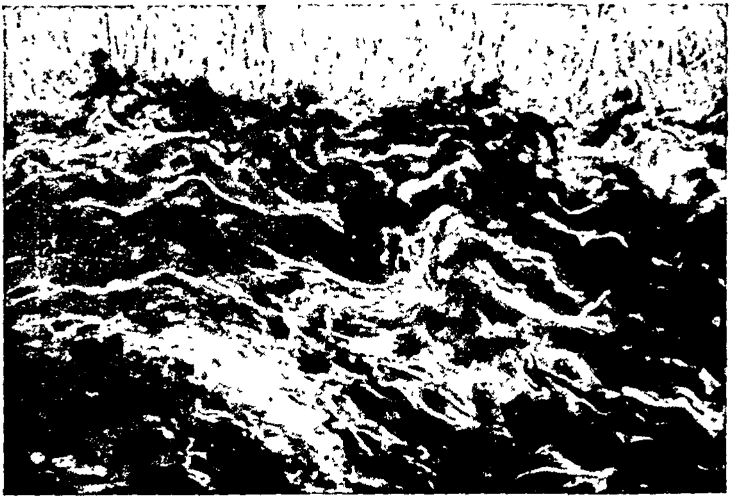

Fig. 15. High power (900X). Biopsy specimen from completely epithelized fenestration cavity showing tightly arranged collagen bundles after the disappearance of intercellular fluid.

Another pain syndrome, established as a result of injury and involving sympathetic effects, has recently been described by Casten and Betcher.[47] They have termed the condition "Reflex Sympathetic Dystrophy" and defined it as "an excessive or abnormal response of an extremity to injury." It is characterized by *1.* excessive and prolonged diffuse pain of varying intensity, seldom limited to a definite nerve distribution; *2.* vasomotor disturbances of the skin, either slight or severe, and consisting of either constriction or vasodilation; *3.* delayed functional recovery, and *4.* atrophic changes. Recommended treatment is interruption of the sympathetic outflow to the extremity, either by continuous procaine ganglion block or by surgical ganglionectomy.

The mechanism of referred pain is not well understood. A consensus of opinion on the subject would seem to be that there is a feedback circuit, possibly following sympathetic pathways along the blood vessels from an established trigger area, through the central nervous system, to the area of referred pain. This feedback mechanism is set off by stimulation of the trigger area and is maintained by the establishment of some hypersensitive state in the sympathetic arcs, causing a continuous discharge of impulses and a constant pain cycle.

In speculating on the mechanism of referred pain in the occasional patient following ear surgery, it seems evident that there is a definite autonomic relationship. Vasoconstriction and vasodilation of the vessels of the face, intermittent blurred vision, the failure of the pain pattern to correspond to somatic peripheral nerve distribution, and the aggravation of symptoms by fatigue and emotional stress tend to confirm it.

The establishment of a trigger area in the cavity also appears logical. A possible explanation is that in those patients where healing in the cavity has been long delayed due to infection, there is repeated injury to the granulation tissue resulting in a greater amount of scar tissue. When the scar tissue undergoes a shrinkage, a trigger area may be established as a result of stretching sympathetic fibers along remaining vessels in the area. The effect of eliciting referred pain by stretching the autonomics is well known. The impression of this pathophysiological explanation for the trigger mechanism is heightened by the observations that the persistence of symptoms of referred pain parallel the period of maturation and contracture in the scar tissue and usually disappear when this

period is ended, presumably with the ingrowth of elast
and gradual resolution of the cicatricial nature of th
At any time during the period of contracture of the
sue, if the epithelium in the new cavity breaks dow
reappearance of infection and reestablishment of incre
tercellular fluid which renders the connective tissue m
itc, the symptoms of referred pain disappear; further
patients who have never complained of postoperative
pain, but who develop a cyst in the cavity, with related
ing of the skin and underlying connective tissue, the s
tern of referred pain will often appear, persisting u
cyst is opened and pressure relieved.

B. *Auditory Changes Due to Cicatrization.*

It has long been observed that patients with chr
hesive deafness have better hearing when the ear is d
ing than when it is dry. In the presence of a disch
connective tissue adhesions are soft and have greater el
When the ear becomes dry these adhesions lose inte
fluid, undergo contracture, and become tight and stiff
ing in restricted movement of the ossicles.

Some variations in hearing observed after fenestra
erations have been difficult to understand. Explanatio
ably are to be found in the pathophysiological change
take place in the connective tissue. It has been obser
the highest hearing level, following a successful fenes
usually is present when the cavity is in the early stages
ing. During this period the pedicle flap over the fen
still relatively thick but very sensitive to sound imp
cause there is a large amount of intercellular fluid in
sues. It is surrounded in the cavity by newly formed
connective tissue (see Fig. 4). If healing takes place
infection, the connective tissue around the pedicle flap
thin, and the effects of contracture are small. There
very slight drop from the maximum hearing level afte
lization takes place. This is due to a reduction of inte
fluid incident to normal maturation changes taking
all healing connective tissue. On the other hand, whe
standing infection occurs in the cavity, there is great
sition of collagen in the deeper layers of connective tis

Fig. 14). When healing finally occurs, a thicker, more sclerotic layer of scar tissue becomes stiff and inelastic, due to the disappearance of intercellular fluid (see Fig. 15). Since sound passing through the pedicle flap overlying a fenestra is influenced by the mobility of that tissue, which in turn is affected by the mobility of the continuous layer of connective tissue in the cavity, there is a drop in hearing. This drop, on the order of 5 to 10 db, is noticeable clinically and audiometrically almost immediately after a cavity has become dry. It is interesting to observe that if the ear becomes reinfected, with consequent return of intercellular fluid to the connective tissue, the hearing will resume its previous level. There are some fenestration patients, with poor healing in the cavity, who experience a drop in hearing even a year or two after the cavity has become dry. This is not always due to bony closure.

There is a final phase of shrinkage in the scar tissue which characteristically takes place one or two years after healing has occurred. This is secondary contracture, which results in further stiffness and diminution of elasticity. It is not necessarily the final fate of such connective tissue, since there is some evidence to indicate that scar tissue actually tends to regress,[48] and that in time elastic fibers grow into the region, restoring to it a certain degree of elasticity.

In the present uncertain state of our knowledge we need to be cautious concerning changes which take place in connective tissue. That changes do take place which have clinical significance and which are influenced by the type of healing, seems evident. These changes in connective tissue which can influence the elasticity of tissue overlying a surgically-made window to the inner ear cannot be ignored in the study of procedures to improve the physiology of hearing in deafened individuals.

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STAPHYLOCOCCUS BACTERIOPHAGE LYSATE AEROSOL THERAPY OF SINUSITIS.

A. ERNEST MILLS, M.D.,*
Medford, Mass.

A fresh approach to the treatment of paranasal sinusitis is presented as a timely adjunct to the problem of infection caused by the antibiotic-resistant Staphylococcus (Micrococcus pyogenes var. aureus). A specific polyvalent bacteriophage lysate is administered by nasal aerosol, thus reaching the areas where this common pathogen is usually found, the anterior nares, as well as the respiratory tract. The paper draws upon the clinical experiences of the author and a number of associates, from the early use of this therapy in 1946 to the present. A fuller appreciation of the immunizing, hyposensitizing, and lytic possibilities of the lysate will help to clarify considerations justifying its use, and to reveal broader fields for clinical investigation.

BACKGROUND OF BACTERIOPHAGY.

After the discovery, by Twort and d'Herelle in 1915 and 1917, of the phenomenon of bacterial lysis by type-specific, parasitic, bacterial viruses, non-pathogenic to man, many different phages were isolated and put to therapeutic trial. Eaton and Bayne-Jones,[1] in a comprehensive review of the clinical data available up to 1934, expressed the opinion that the results were conflicting and difficult to assess. Krueger and Scribner[2] carried forward this cumulative survey to 1941, and their conclusions were equally critical; however, both papers called attention to the continuing favorable experiences with Staphylococcus bacteriophage, and thought it worthy of further study.

At about the same period, the therapeutic possibilities of this specific phage were strongly supported by MacNeal,[3]

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whose knowledge of, and confidence in the product, is reflected by his statement, "By the skillful use of bacteriophage, low-grade infections often are rendered trivial, acute infections may be aborted, deep-seated lesions may heal without drainage, and desperate conditions may be caused to heal in a surprising manner". He generally used and recommended intravenous administration. Other methods in vogue included the usual subcutaneous or intramuscular injections, wet dressings or sprays for superficial lesions, tamponades, inunctions, instillations or irrigations in body cavities and openings, and oral dosage for treatment of the gastrointestinal tract.

Herein is reported a technique for the aerosol administration of staphylococcus bacteriophage lysates, devised by the late Robert E. Lincoln, M.D., in 1946, for the treatment of infections involving the sinorespiratory tract. Admittedly, his presentation of the subject to the medical profession was incorrect; nevertheless, this paper is an attempt to overcome prejudice and to suggest a valuable contribution to medicine.

COMPOSITION AND PREPARATION.

Two staphylococcus bacteriophage lysates are prepared by the Lincoln Foundation Laboratory in Swarthmore, Pa. Primarily, the phage was the same in both; identified as Gratia B-985—from a stock collection maintained at the Boston University School of Medicine—and was selected because of its high polyvalency. As host cells for preparing the cultures, two strains of toxigenic staphylococci are used.

The A-1 lysate (Alpha) is prepared from a parent culture of S. aureus (Lincoln strain No. 1), originally obtained from the nasal passages of a patient with acute sinusitis and infectious diarrhea. The B-7 lysate (Beta) is made from a parent culture of S. aureus (Lincoln strain No. 7), first taken from a case of sinusitis with secondary infectious hepatitis. Both strains possess all the recognized characteristics of virulent toxigenic micrococci; they are coagulase-positive, produce exotoxins, alpha hemolysins, polysaccharides and penicillinase. The organisms are grown in beef-heart infusion broth and lysed by adding staphylococcus bacteriophage. When lysis is complete, the resulting solution is sterilized by filtration. To insure viability of the phages, a preservative is not added. They retain full potency for at least three months, and keep well under ordinary refrigeration.

The final product has an extremely high lytic activity (phage content of 10^{-8} to 10^{-10}), and must pass all tests for sterility from bacteria, safety and absence of toxicity, in conformity with governmental requirements. Laboratory procedures are carried out under expert supervision by competent personnel, experienced in the preparation and quality control of biologics.

ADMINISTRATION AND DOSAGE.

For the treatment of sinusitis, staphylococcus bacteriophage lysate is administered as an aerosol by nasal inhalation. The mist is formed by releasing compressed air or oxygen into a Vaponefrin nebulizer, and is delivered into the nares through a Y tube with glass nasal tips. It is usual to start treatment with the inhalation of .25 cc. of the Alpha lysate. Subsequent doses are gradually increased at one to three-day intervals, until the full maintenance of 1 cc. is reached. If progress is not continuous, the Beta lysate may be added for fuller effect.

It is emphasized that determination of dosage and the interval is highly individualized, depending upon the post-treatment reactions. Patients are told they may have transient episodes of malaise, especially during the early stages of the therapy, that occur as a rule, about four to six hours after an inhalation. They vary from fatigue with mild aches and pains, to a more severe type with chills followed by slight fever of short duration, grippe-like symptoms and more marked general discomfort, easily relieved by medication. There may be temporary reactivations of any previous illness, due to sensitization phenomena. Reactions, which last from a few minutes to a few hours, tend to disappear as treatment progresses and are succeeded by a feeling of well-being. The more uncomfortable ones indicate a reduction in the amount of phage given, temporarily, or a lengthening of the treatment interval. Post-treatment effects are due, in part, to local bacteriolysis, with a release of toxins which stimulate the production of specific antibodies, and to a bacterial desensitization.

The aerosol therapy should be continued until reactions cease, or till relief from all symptoms, objective and subjective, is realized. When definite alleviation has been established, a monthly maintenance or booster dose tends to forestall and prevent relapses and to lessen the number and the severity of head colds.

ADJUNCTIVE MEASURES.

During the course of treatment, daily nasal irrigations with warm dextrose solution are advisable to clear the passages of mucus and crusts, and to prepare the mucosa for the absorption of the lysates. Specific written instructions are issued to the patient for this home procedure. If diarrheal symptoms accompany the sinusitis, the Alpha lysate is used orally in 1 to 2 cc. dosage in 30 to 60 cc. of water. Taken with one-fourth to one-half grain (.015 to .030 gm.) codeine sulfate, the phage has proven to be an exceptional aid in combatting the infectious diarrheas, so often present in such conditions, and in staphylococcal enterocolitis.

The usual supportive measures, as a diet rich in protein, adequate rest, prevention of calcium and vitamin deficiencies, and relief of nervous tension, are requisite. Other hyposensitizations, the use of antihistamines and antibiotics orally or parenterally, in no way inhibit the action of the bacteriophages, which should be looked upon as an auxiliary to other necessary therapy.

CLINICAL OBSERVATIONS.

The results of staphylococcus bacteriophage lysate aerosol therapy in 60 cases of chronic sinusitis are summarized. They constitute a random sampling of patients who had proven stubborn to conventional treatment, and include 26 males and 34 females. The youngest was six years of age, the oldest, 70, and the average age for the group was 40. The number of aerosols ranged from five to 90 per patient, with an average of 18 treatments. In 27 cases (45 per cent) the results were rated as excellent, in 20 (33 per cent) good, in 10 (17 per cent) fair, and in three (5 per cent) poor. For the combined groups of good to excellent, the percentage is 78 per cent.

In agreement with clinical observations made at the Lincoln Foundation clinic, physicians using the phages report numerous cases that had been resistant to all other treatment made satisfactory improvement within two to six weeks, or after 10 to 20 inhalations. Intrafamilial cross infection must be considered in the recurrent case under treatment, and contacts should be advised to have the therapy, even though they may not be ill due to a high level of antibody production.

CASE REPORTS.

The following case is of special interest because it presents a severe staphylococcal infection of the nasopharynx, with membrane formation, super-imposed on and masking a chronic sinusitis.

Case 1: E. D., a six and a half-year-old girl was seen by the author on an emergency call the early morning of Aug. 6, 1953, because of nausea, vomiting and a severe croupy cough of three days' duration. At age six, the onset of sinusitis had been noted, with frequent colds, constant post-nasal drainage, poor appetite and lack of vigor. Four attacks of tonsilitis had occurred since March, 1953, coincident with acute exacerbations of the sinusitis. Swollen cervical glands were present with each illness and did not subside entirely afterward. July 21, she became sick with a sore throat, stuffy nose, a temperature of 101° F. and severe lassitude. Bilateral cervical adenitis developed rapidly. The family physician was called and gave 300,000 units of procaine penicillin, intramuscularly, and 400,000 units by mouth, on two successive days. He then went on a scheduled vacation.

The child was frail, toxic, and orthopneic with stertorous breathing. The lips and fingers were moderately cyanosed, this condition having become more noticeable to the mother during the night. Temperature was 101° F., pulse 120 and weak. A thick grayish-white membrane, which bled on removal, covered the fauces, tonsils, pharynx, and extended upward into the posterior nares. The nasal passages were blocked by this same membrane. Because of the emergency, 40,000 units of diphtheria antitoxin were given, though immunizations had been received at the age of one year, with a booster at age three. Procaine penicillin, 1,000,000 units, was administered intramuscularly once only, because she developed a reaction to it. Syrup of ipecac induced vomiting and expectoration of considerable phlegm, with great relief from the stridulous breathing. Steam inhalations, containing Alevaire, were kept going constantly for the first 24 hours. Diphenhydramine (Benadryl) 25 mg. was given orally for its sedative and antihistaminic effect. Alevaire was also used by mouth as an aerosol, to loosen mucus.

Two cultures of the nose and throat showed a pure growth of S. aureus. After douching the passages with warm dextrose solution, 1 cc. of the Alpha bacteriophage was administered by nasal aerosol and inhaled orally, 1 cc. of the Alpha lysate in 30 cc. of water was taken by mouth, because of diarrhea, present two days. The phage lysate was given once a day nasally and orally, for 11 days, then on alternate days for four doses. At this time, the family physician returned, and the treatments were discontinued.

Within 24 hours after starting the phage therapy, the child was resting comfortably, with no orthopnea or cyanosis. Fluids were taken easily, and in 36 hours soft solids were added. The diarrhea subsided in 48 hours and the stools became normal. The tenacious membrane sloughed off with remarkable ease and in three days had entirely disappeared. The cervical adenitis seemed to melt quickly, noticeably decreasing within 24 hours, and in one week was gone. Interest in surroundings and play returned within 48 hours with a rapid lessening of toxicity. Post nasal discharge and other symptoms of sinusitis cleared within one week, the only residual sign was a mild anemia and the girl was apparently well. Two months later it was learned that there were no after effects, further treatment had not been required, no sinusitis or adenitis was present and she had gained six pounds in weight.

The average case of sinusitis with bacterial allergy is illustrated by the following report from A. G. Baker, M.D., Ridley Park, Pa., and is used here with his permission.

Case 2. J. H. W., a business executive, age 45, came under Dr. Baker's care Sept. 30, 1952, complaining of copious post nasal drip, severe headaches on occasion, morning cough and increasing difficulty with hearing. Onset occurred early in 1950, with an acute attack of sinusitis. Previous treatments by otolaryngologists included surgery for correction of a deviated septum and to provide a maxillary window. These and the usual ear, nose, and throat regimen gave only temporary relief. The diagnosis of sinusitis was confirmed, both frontal and maxillary tenderness being present. Cultures from the nose yielded a growth of S. aureus with some hemolytic streptococci. The hemoglobin was 84 per cent, sedimentation rate 8 mm. in one hour, RBC and WBC totals normal. Neutrophiles were low (34 per cent) while lymphocytes were increased (62 per cent). There was no increase in eosinophiles though skin tests showed moderate reactions to house dust, orris root and feathers.

Staphylococcus bacteriophage lysate aerosol therapy was started on Oct. 6, 1952. Inhalations were given daily to October 21 and then at two to five-day intervals until December 6—a total of 23 treatments in eight weeks. Concurrently, a course of six hyposensitizing injections (house dust extract and a serobacterin) was given at intervals of a week or more. The patient experienced reactions after a few of the early aerosol treatments, some were mild grippe like symptoms, others were more severe but of short duration with chills, slight fever and aching joints. Within five days there was general improvement, with less post nasal drainage, the cough had stopped entirely and sinus tenderness had abated. Further gradual relief followed, not only in the sinusitis but his hearing also was definitely benefited in ten days and after four weeks was reported "perfect." He was asymptomatic by December and bacteriophage treatments were discontinued after Dec. 6, 1952. A follow up inquiry in late 1954 noted that he was feeling fine and was free of sinus trouble.

DISCUSSION

Latent Infection

While the literature on sinusitis is voluminous and often confusing, nevertheless, there is general agreement on many basic points.[4] It is acknowledged that normally the nasal mucosa harbors a mixed flora of pathogenic and non-pathogenic organisms. In health these are apparently harmless, being held in check by the combined effects of ciliary action, the slightly acid pH of nasal secretions, the lysozyme present in mucus, and a balanced antigen/antibody ratio. Vital resistance may be lowered by physiologic disturbances or stress: exhaustion from previous disease, trauma, surgery, shock and old age; mental or physical fatigue and menstruation; malnutrition, vitamin deficiencies and the extended use of the broad-spectrum antibiotics; prolonged nervous tension, and especially by exposure to extremes of wet and cold weather, high humidity having the greatest influence.

The foregoing provoking factors favor the growth of these dormant microorganisms; virulence increases, invasion of tissues and infection of sinuses take place. Interference with ventilation and drainage causes a toxic absorption, which is reflected in pain and malaise, with lassitude, weakness and general ill feeling.[5]

Many references are in accord that the staphylococcus is the pathogen most frequently isolated from the nose, often dominant and probably the most virulent.[6-9] Welch[10] states, "The organism is ubiquitous and probably is the cause of more infections in man (not excluding the common cold) than any other known microorganism".

Bacterial Allergy.

Similarly, there is a general agreement that, "70 to 80 per cent of all cases of chronic sinusitis have allergy as their basic pathology. There is no doubt but that bacterial infection of the respiratory tract may cause hyperplastic sinusitis and asthma".[11] Allergy, particularly bacterial allergy, plays an important role in nasal and sinus disease, because the resulting edema and hyperplasia of the mucosal layers close the ostia, block drainage, impair ventilation and ciliary action, favor infection and build up internal pressure, which in turn increases toxic absorption. In the majority of cases of sinusitis, the process is one of allergy combined with infection.[4, 12-16]

Swineford and Holman,[17] reporting on a large series of cutaneous reactions to the polysaccharide and nucleoprotein fractions of 14 different bacteria, found that skin tests for bacterial allergy gave both immediate and delayed reaction types. The larger number, and the higher percentage of the immediate reactions, were elicited by the polysaccharides of S. aureus. Delayed or tuberculin-type reactions also were frequently induced by the nucleoproteins of the same organism. In later studies on bacterial allergy, Swineford,[18] using pneumococcal polysaccharide experimentally on passively sensitized guinea pigs, demonstrated that, "Desensitization may be readily produced by specific hapten aerosol". This study has a direct and important bearing on the sinusitis problem and favors the assumption that staphylococcal polysaccharides and nucleoproteins in the bacteriophage lysates may act as specific antigens in the bacterial allergy associated with this disease. The supposition requires further laboratory and clinical research for its corroboration.

Baker,[19] in a series of 60 cases of chronic bronchial asthma and sinusitis treated by aerosol inhalation of these phage lysates, found the therapy afforded sustained symptomatic relief in every instance, when used as an adjunct to other desensitization measures. No evidence of toxicity, local irritation, or anaphylactic-type reaction was apparent during the course of treatment. In a series of six asthmatic patients, he gave aerosols with the culture medium alone, without relief.

Staphylococcus Toxin and Vaccine Action.

Lucic[20] and Burky[21] were able to show that substances with mild antigenic properties might become activated if combined with staphylococcus toxin. Also, Blair[22] states, "In common with some other pathogenic bacteria, staphylococci produce a spreading factor (Duran-Reynals, 1942) which, by increasing the permeability of connective tissue, aids in the initial formation of the local lesion. The spreading factor is related to the mucolytic enzymes, or hyaluronidase, and is similar to a spreading factor found in testicle and other body tissues. . . . Experimental infections with other bacteria or with viruses are enhanced by staphylococcic spreading factor."

According to Larkum,[23, 24] a superior vaccine is prepared by lysing an organism. Lytic filtrates possess an antigenic capacity, in that they induce specific neutralizing antibodies, which have many of the attributes of an antitoxin. It is the bacteriophage itself which has this property. Phage combines with strain-susceptible bacteria, and initiates a change which progressively renders them more amenable to phagocytic ingestion. The more potent the phage, the greater this action becomes.

Formerly, bacteriophages were utilized mainly by injection, and the system, perforce, formed antiphage (antilysin) which gradually prevented phagic lysis to a degree. When used as an aerosol, intranasally, the development of antiphage is slowed as the mucosal lining of the sinorespiratory tract has a chance to select or reject fractions of the lysate.

Mucous Membrane Absorption.

As a means of drug administration, the nasal mucous membranes permit the passage of many antigens, serums, toxins, toxoids, pollen extracts, vitamin B_{12} and others. Chait and Walzer[25] have shown that this absorption is fast. Tonndorf et al.,[26] in testing various drugs to combat motion sickness, found that absorption in this area was more rapid than after oral administration, that it approached more nearly the speed of subcutaneous injection, and that a smaller dose was required than by mouth. Ease of administration and rapidity of absorption have been repeatedly emphasized. Clinical observations indicate that this is equally true of the phage aerosols used in this study.

COMMENT.

Bacteriophages make a relative immunity, in which the recovery is not complete. This active resistance to infection, or non-sterile immunity, is characterized by the fact that the antibodies produced are sufficient to suppress the majority of susceptible pathogens, but leave a few resistant organisms, which are subsequently able to repeat the infection, under conditions of physiologic stress. This is the reason for maintenance or booster treatments. Regardless of the varying phage sensitivity of staphylococci, the active immunization value of the lysates is based upon their close antigenic relationship.

All available staphylococcus phages (used for classification purposes) have been tested for lytic ability, and none compare with the high polyvalency exhibited by these preparations. Phage resistance has not occurred among many strains of micrococci tested in the laboratory. The effectiveness of these bacteriophages in therapy is in no way limited by the antibiotic resistance of the prevailing strains of S. aureus.

The basic premise for the use of bacteriophages in general was that bacterial viruses on parenteral injection, would lyse their specific host cells anywhere in the body. It is now realized this occurs only on actual contact, and the good results previously obtained were due to a specific immunizing action, an increase in susceptibility to phagocytosis and to a hyposensitizing effect.

The application of the lysates, by nasal aerosol, was originally chosen as an effective method of treating sinusitis. Through clinical observation of a large number of patients thus afflicted, it was noted that certain collateral symptoms, or concomitant disease entities, were also improved or eliminated as the foci of infection in the paranasal sinuses were cleared. These observations will be the topic of a future paper.

SUMMARY AND CONCLUSIONS.

While the use of strain-specific bacteriophage lysates or filtrates is not new, the emphasis in this report is on their administration by nasal aerosol inhalation for the treatment of paranasal sinusitis. The earlier literature refers to the use of phages in the form of nose drops, packs and irrigations as a means of local application to the nasal passages and sinuses. Reviews are conflicting as to their value, but do call attention to the continuing favorable experiences with staphylococcal types.

Clinical observations of patients having chronic sinusitis, treated with staphylococcus bacteriophage lysates prepared by a special method, show gratifying results in 78 per cent of cases. These were stubborn conditions that had not been relieved by a conventional regimen. Oral use is an exceptional aid in combatting infectious diarrhea, commonly found with sinusitis, and in staphylococcal enterocolitis.

The preparation, administration and dosage are outlined. The aerosol is given by standard intranasal technique under a pressure of five to six liters of air or oxygen per minute, thereby reaching all the available surfaces of the nasal mucosa, sinuses, bronchi and the lungs on inhalation. In this manner, an effective local concentration of the bacterial viruses is made to the areas where the principal pathogen, staphylococcus aureus (micrococcus pyogenes var. aureus), is most numerous; the anterior nares being the chief reservoir of these potentially virulent microorganisms in health and in disease. Latent infection in the paranasal sinuses, activated by various physiologic disturbances or stress factors, may be the site of parenteral introduction of bacterial protein with a consequent sensitization of the body tissues. Staphylococcal spreading factor may enhance infection with other bacteria or viruses.

The rationale for the use of staphylococcus bacteriophage lysate by nasal aerosol and oral dosage takes into consideration certain advantages and potentialities that reveal broader fields for clinical investigation:

The bacteriophage used in preparing the biologic is a well established and actively polyvalent strain that has a lytic effect over a wide spectrum of pathogenic and non-pathogenic staphylococcal groups.

The phage is antigenic; mucous membrane absorption produces a more effective and rapid active immunization than the injection of an orthodox vaccine. The selective powers of the nasal mucosa minimize the possible development of antiphage (antilysin).

The lysate is anti-allergic; certain constituents, such as polysaccharides and nucleoproteins, act as haptens or partial antigens, and bring about a gradual hyposensitization to micrococcal bacterial allergy, which is present in 70 to 80 per cent of sinusitis cases.

The immunizing and hyposensitizing results are exerted against all strains of micrococci because of their close antigenic relationship, regardless of their varying sensitivity to phagic lysis The development of resistance to the lysates has not occurred in laboratory tests

Staphylococcus bacteriophage lysates administered by nasal aerosol and oral technique are stressed as a valuable adjunct to the treatment of the antibiotic-resistant micrococci

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309 High Street.

ANNUAL ASSEMBLY IN OTOLARYNGOLOGY.

The Department of Otolaryngology, University of Illinois College of Medicine, announces its Annual Assembly in Otolaryngology from October 1 through 7, 1956. The Assembly will consist of an intensive series of lectures and panels concerning advancements in otolaryngology, and evening sessions devoted to surgical anatomy of the head and neck, and histopathology of the ear, nose and throat.

Interested physicians should write direct to the Department of Otolaryngology, 1853 West Polk Street, Chicago 12, Ill.

ABLATION THERAPY FOR THE RELIEF OF MENIERE'S DISEASE.*†

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A sudden attack of severe vertigo, accompanied by vomiting, can be a terrifying experience. When the episodes in Ménière's disease recur frequently the patients become very apprehensive for fear of having an attack while at work or on the street. At this stage they are incapacitated, and quite willing to make some sacrifice to acquire relief of symptoms.

The diagnostic criteria for Ménière's disease include tests for auditory and vestibular function. It is these tests, in fact, which make it possible to differentiate Ménière's disease from other vertiginous disorders. The criteria may be listed as follows:

1. Attacks of vertigo and vomiting.

2. Perceptive type hearing loss, manifested by threshold loss for bone and air conduction.

3. Loudness recruitment in the involved ear.

4. Diminution of caloric response in the involved ear.

These criteria must be met before ablation therapy can be advised. The classical symptoms of Ménière's disease are well-known to all of us, and need no further description at this time.

It is not my purpose to discuss the medical management ot this disease. We all recognize that medical management is fairly successful in many patients, and it should be used first,

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† This is a summary of the studies completed to date and the conclusions reached. A complete report is in preparation.

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resorting to ablation methods only if vertiginous attacks continue to incapacitate the patient.

The mechanical receptors for both the vestibular and auditory systems operate on hydrodynamic principles and have attained, therefore, an intimate anatomical relationship in the labyrinth to the temporal bone. For this reason surgical attempts to ablate the vestibular system and leave the auditory structures intact have not been uniformly successful; nonetheless, there are several procedures which have as their object the selective destruction of vestibular function and preservation of hearing. Two methods should be mentioned. *1.* There are reports from Italy of good selective destruction of the vestibular system with a technique utilizing high intensity ultrasonics.[1] To my knowledge it has not been used in this country. *2.* By sectioning the vestibular nerve it is possible to preserve pure tone thresholds at the preoperative levels in some patients. To my knowledge, however, there has been no evaluation of speech discrimination following vestibular nerve section. Testing of speech intelligibility is particularly important where there is possibility of injury to the cochlear nerve. It is now fairly well established that a decrease in nerve fibre population to the cochlea results in a loss of auditory discrimination out of proportion to the loss for pure tone thresholds. Following vestibular nerve section I would like to know, not only the level of pure tone thresholds, but also the speech discrimination scores. Has practical hearing been preserved?

Before labyrinth ablation for unilateral Ménière's disease is undertaken the following criteria should be met:

1. The diagnosis should be established conclusively through history and characteristic auditory and vestibular findings.

2. The symptoms must be at least moderately incapacitating.

3. The auditory function of the opposite ear must be good without evidence of progressive disease.

TRANS-TYMPANIC LABYRINTHOTOMY.

Trans-tympanic labyrinthotomy is the simplest effective surgical method of destroying inner ear function. The tech-

Fig 1 Surgical procedure for trans tympanic labyrinthotomy See text for explanation

nique varies slightly from the original trans-tympanic technique described by Lempert[2]

An endaural incision is made as for the stapes mobilization, and the middle ear is exposed (see Fig 1). The incudo stapedial joint is disarticulated by displacing the long process forward. The stapes tendon is cut with an endaural scissors. The head of the stapes is engaged with a needle and with gentle manipulation it is loosened. A hook is then introduced into the intracrural space and the stapes removed. A small barbed instrument is introduced through the round and oval windows and manipulated so as to disrupt the membranous labyrinth. A suction tube is introduced through the oval window and the contents of the vestibule aspirated. Small pieces of gel-foam are placed in the vestibule to stimulate connective tissue proliferation and acquire thereby a firm barrier between the middle ear and subarachnoid space. The operating time is about 20 minutes.

TABLE I. TRANS-TYMPANIC LABYRINTHOTOMY.

Case	Method	Caloric	Hearing	Recurrence	Follow Up
1	Opened	Lost	Lost	No	19 mo.
2	Windows	Decr.	Lost	Yes (7 mo.)	19 mo.
3	Only	Lost	Lost	No	18 mo.
4		Decr.	Lost	Yes (12 mo.)	18 mo.
5	Probed	Lost	Lost	No	14 mo.
6	and	Lost	Lost	No	14 mo.
7	Aspirated	Lost	Lost	No	11 mo.
8	Inner	Lost	Lost	No	10 mo.
9	Ear	Lost	Lost	No	9 mo.
10		Lost	Lost	No	8 mo.
11		Lost	Lost	No	3 mo.
12		Lost	Lost	No	3 mo.
13		Lost	Lost	No	2 mo.

During the past 19 months this operation has been performed on 13 patients with incapacitating unilateral Ménière's disease. The operative procedure in the first four patients consisted only of removing the stapes and rupturing the round window membrane without probing and aspiration (see Table I). In two of these a mild caloric reaction to ice water remained in the operated ear after surgery. Both patients had recurrence of dizzy spells, although the attacks were less frequent and less severe than before surgery. A second operation including probing and aspiration of the inner ear resulted in total loss of caloric reaction and relief of symptoms. There was total loss of caloric reaction to ice water and relief of dizzy spells in all patients in whom the contents of the vestibule were probed and aspirated. The average age of these 13 patients was 53 years, average hospital stay seven days, average duration of symptoms prior to operation four and one-half years, and the average time lost from work after operation 23 days.

Fig 2 Intra-tympanic steptomycin therapy is given through tubing which is introduced into the middle ear through a knife wound made through the annulus tympanicus

INTRA-TYMPANIC STREPTOMYCIN.

An equally effective method of accomplishing unilateral labyrinthine ablation is intra-tympanic streptomycin therapy. The drug is injected through small plastic tubing which has been introduced into the middle ear through a small knife wound in the annulus tympanicus (see Fig. 2). This tubing has an outside diameter of .61 mm. and inside diameter of .28 mm.* The tubing has been left in place as long as seven days without discomfort, and leaves not even a temporary perforation after removal. By varying the frequency of injection and the concentration of the streptomycin solution it was hoped that a method might be developed whereby vestibular function could be destroyed and hearing saved. The results showed that successful treatment depends upon abolishing caloric reaction to ice water (see Table II). When this was accomplished hearing was also lost. The first three patients received inadequate therapy; the caloric responses were unchanged or slightly decreased, and recurrences followed. Of the five cases receiving adequate therapy the longest follow-up was only seven months, but in view of the total loss of ice water caloric reaction in the treated ears, a recurrence of symptoms seems unlikely. The results suggest that the treatment may be most effectively accomplished by injecting about .1 cc. of streptomycin every four hours in a concentration of .5 grams per cc. in physiological saline, with injections being given for one day beyond the stage when vestibular symptoms are fully developed. That would be about four to five days. The vestibular reactions and after-effects of treatment are identical to those following labyrinthotomy. The procedure has one distinct advantage over labyrinthotomy in that a general anesthetic is not required.

TABLE II. INTRA-TYMPANIC STREPTOMYCIN.

Case	Gm./cc.	Days Therapy	Caloric	Hearing	Recurrence	Follow Up
1	.5	1	Same	Same	Yes	20 mo.
2	.5	2	Same	Same	Yes	20 mo.
3	.5	2	Same	Same	Yes	10 mo.
4	.5	3	Lost	Lost	No	7 mo.
5	.5	4	Lost	Lost	No	5 mo.
6	.125	7	Lost	Lost	No	4 mo.
7	.125	6	Lost	Lost	No	3 mo.
8	.125	7	Lost	Lost	No	3 mo.

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* Polyethylene tubing, size PE-10, Clay-Adams Co., Inc., 14 E. 25th St., New York 10, N. Y.

In an experiment in which a number of cats were given intra-tympanic streptomycin it was found that there was a total loss of hair cells in both the vestibular labyrinth and cochlea (see Fig. 3).

PARENTERAL STREPTOMYCIN THERAPY.

The treatment of patients with bilateral Ménière's disease is a different problem, for here it is imperative that all existing auditory function be preserved. This is best accomplished by parenteral streptomycin therapy, by which it is possible to selectively destroy the vestibular function in both ears and save hearing. Previous reports of moderate success from the use of streptomycin for Ménière's disease have been made by Fowler,[3] Hamburger et al.,[4] Ruedi[5] and Hanson.[6] My experience with the drug has more or less paralleled theirs.

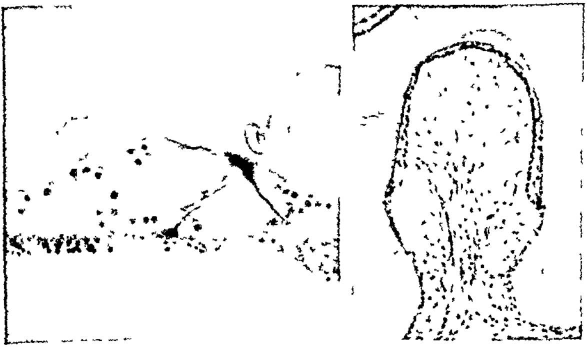

Fig. 3. Organ of Corti and crista ampullaris of a cat given one injection into the auditory bulla of one half cc. of physiological saline containing 0.25 gram of streptomycin sulfate. There was a total loss of hair cells in the inner ear.

Several of the first patients received three gm. per day for part of the treatment period, but in this dosage the onset of toxicity was too precipitous to allow for satisfactory control of the end result. The ideal quantity was considered to be 2 gms. per day, given intramuscularly in two equally divided doses, for this resulted in a more gradual development of toxic effect with the desired end-point being reached in two to four weeks. The treatment was given for seven to ten days on an out-patient status, after which the patients were admitted to the hospital where more careful observation was possible.

The importance of frequent auditory and quantitative caloric testing cannot be over-emphasized. I found no advantage in routinely performing both hot and cold caloric tests. The minimal caloric tests were used, which were performed by injecting 5 cc. of water at 80° F. against the postero-superior quadrant of the eardrum at a rate of ½ cc. per second, and observing the nystagmus through illuminated Frenzel glasses in a darkened room. The reaction time was recorded from the end of injection to the end of nystagmus. If there was no reaction to 80° F. water, then 5 cc. of ice water were used. When spontaneous nystagmus existed, the ear on the side opposite the direction of the quick component was tested with water at a temperature of 118°.

TABLE III. PARENTERAL STREPTOMYCIN.

Case	Therapy Days	Calorics			Recurrence	Follow Up
		Right	Left			
UNILATERAL						
1	21	Decr.	Lost	†	No	57 mo.
2[1]	7	Lost	Decr.	††	No	45 mo.
3	36	Decr.*	Lost	††††	No	37 mo.
4	26	Lost*	Decr.	††	No	29 mo.
5	14	Decr.	Lost*	†††	No	28 mo.
BILATERAL						
6	24	Decr.*	Decr.*	†††	No	34 mo.
7	12	Decr.*	Decr.*	††††	No	14 mo.
8	15	Lost*	Lost*	††††	No	12 mo.

[1] Given 3 grams per day. Decr. Decreased. * Diseased ears.

For the five patients with unilateral Ménière's disease the streptomycin was discontinued on the first day the ice water caloric failed to give a response in the diseased ear. Four of these patients experienced a total loss of ice water caloric reaction in the diseased ear with some preservation in the normal ear (see Table III). The three patients with bilateral Ménière's disease were treated with the objective of preserving some function in one or both ears. This was accomplished in two patients. A remarkable result was the sustained improvement in auditory thresholds in five of the eight cases. This improvement was not important for the unilateral cases,

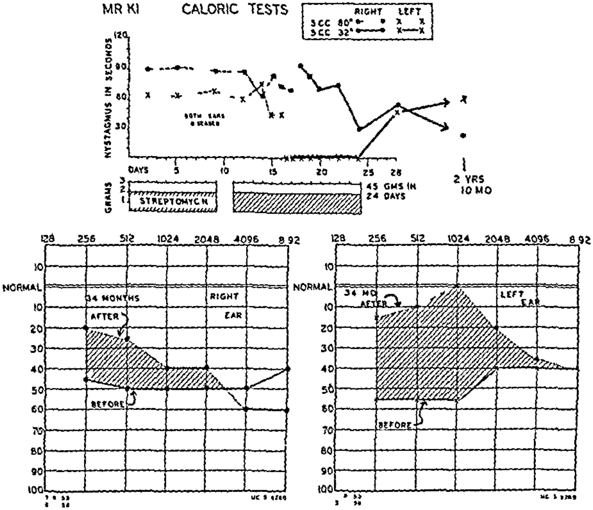

Fig 4 Above Results of minimal caloric tests of patient with bilateral Meniere's disease The tests were performed by injecting 5 cc of water against the eardrum and measuring the time lapse from end of injection to end of nystagmus Note that 80° F water was used for 16 days after which it was necessary to use ice water to get a response The intramuscular streptomycin injections were discontinued on the 24th day when there was no reaction to ice water in the left ear and a mild response in the right ear Two years and ten months later a slight caloric response to ice water remained in both ears He has had no further dizzy spells

Below Audiograms showing remarkable recovery of thresholds The results were confirmed by multiple tests on different days both before and after treatment

but for one patient with bilateral Ménière's disease the remarkable improvement in auditory thresholds was most gratifying (see Fig. 4). The results indicated that the patients in whom some vestibular function was preserved experienced less ataxia and yet had an equally successful rel tiginous attacks. The greatest problem encountered in parenteral streptomycin treatment was prolonged ataxia. The patients with the greatest loss of caloric response had the most severe ataxia. All eight patients treated by this method were discharged from the hospital within one week following treatment. Ataxia was moderately severe for two to three months in all cases. Three were unable to perform normally in their occupations for nine to 12 months. Most were able to drive their autos in two to three months, although one patient required ten months. They all returned to work within periods of time varying from two to nine months. All patients have been completely relieved of vertiginous attacks, the longest follow-up being four years, nine months. Three of eight patients have had attacks of tinnitus and fullness in the ears simulating the auditory symptoms existing prior to treatment. The undesirable symptoms coincident with total loss of vestibular function subsided gradually, so that after one to two years the patients were essentially asymptomatic.

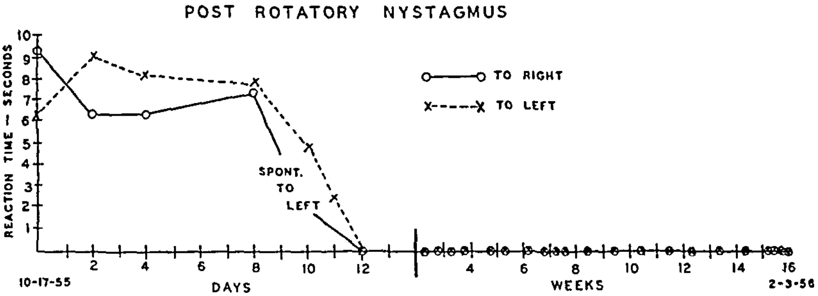

Fig. 5. Post-rotatory nystagmus plotted against time for a cat receiving 200 mgm. per kg. of streptomycin injected intramuscularly in two equally divided doses. Post-rotatory nystagmus was measured from the end of rotation (10 turns in 10 seconds) to the end of nystagmus. Streptomycin was discontinued on the tenth day. Post-rotatory nystagmus was absent on the twelfth day, and no reaction could be elicited during the following two months. On the tenth and eleventh days there was spontaneous nystagmus to the left, so that the turning test to the left was omitted. Auditory thresholds for the experimental ear (right), as determined by the behavioral method,[7] remained normal throughout. The function of the left ear was destroyed by a surgical procedure four months before the beginning of the experiment.

Parenteral streptomycin therapy is not recommended for unilateral Ménière's disease because an equally effective result

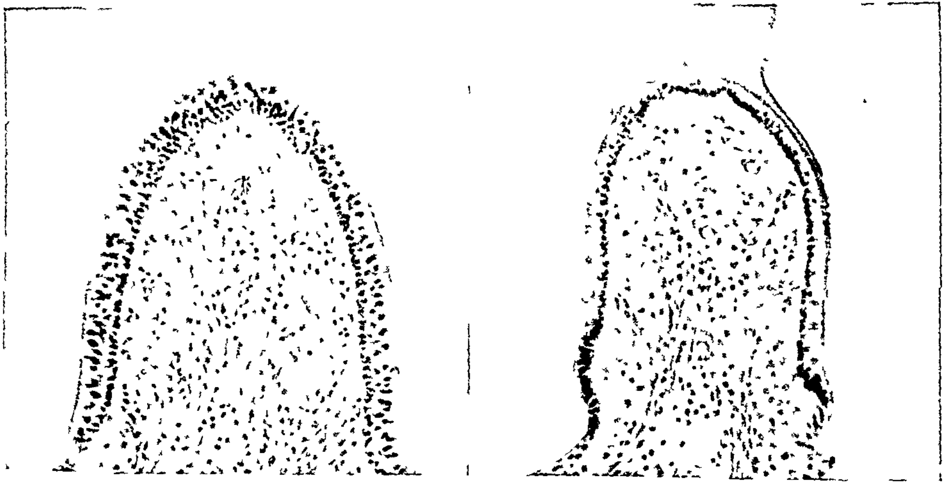

Fig 6 Left Normal crista ampullaris from an untreated control cat
Right Loss of hair cells in crista ampullaris of cat receiving intramuscular streptomycin whose nystagmogram appears in Fig 5 The utricular and saccular sensory epithelium were similarly degenerated The organ of Corti was normal throughout

can be accomplished by unilateral labyrinth ablation with less subsequent vestibular upset.

Preliminary studies of an experiment in which streptomycin was given parenterally to cats has revealed that vestibular function can be destroyed and hearing preserved with a good margin of safety. Pathological study has revealed destruction of the vestibular sensory epithelium and preservation of a normal organ of Corti* (see Figs. 5 and 6).

I gratefully acknowledge the generous help of Dr. Roderick C. Davison, of Winnipeg, and Mrs. Rosemary Doran, Dr. Joyce White and Dr. T. Manford McGee, of Detroit, with the animal experiments.

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6. HANSON, H.: The Treatment of Endolymphatic Hydrops (Meniere's Disease) with Streptomycin. *Ann. Otol., Rhinol. and Laryngol.*, 60:676-692, Sept., 1951.

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* The results of the animal experiments on streptomycin ototoxicity are incomplete, and the findings presented here should not be interpreted as conclusive. The question of possible streptomycin damage to the brain is also under investigation.

PAPILLOMATA OF THE LARYNX.*

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Perhaps no other laryngeal condition has given rise to greater diversity of conflicting observations and conclusions than has papilloma of the larynx. A study of the literature throws surprising light on current theories of etiology and methods of treatment.

HISTORICAL BACKGROUND.

Surprisingly enough, the condition was recognized long before the invention of the laryngoscope: sometime during the Seventeenth century Marcellus Donalus wrote of laryngeal growths describing them as "warts on the throat."[1]

When, early in the nineteenth century, the Imperial Government of France offered a prize for the best study of croup, one far-reaching result was the considerable impetus given to the investigation of laryngeal growths.[2] The first case of papilloma of the vocal cord reported in the United States was published by Cheesman of New York, in 1817;[3] the patient died without relief. Two decades later Trousseau and Belloc published their review of seven cases, the total reported up to that time.[4] In 1850, Ehrmann published a complete monograph on growths of the larynx,[5] but how many of these were papillomata we are unable to say.

A landmark was reached in 1866, when Elsberg published the first description of the microscopic appearance of laryngeal papilloma,[6] but use of the term "papilloma" may be sus-

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[1] Selwyn-Brown: The Physician Throughout the Ages, Vol. II, pp 455-456. For full data see Bibliography appended.

[2] Mackenzie: Essay on Growths in the Larynx, pp. 2-5

[3] Broyles: Trans. Am. Acad. Ophth., Jan.-Feb., pp. 247-253, 1948.

[4] Selwyn-Brown, op cit., p 457.

[5] Mackenzie: op. cit., p. 5.

[6] Selwyn-Brown: op. cit., pp. 455-457.

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pect until at least 1871, when benign growths of the larynx were first differentiated and classified in Mackenzie's report of 100 collected cases.[7]

RISE OF CONFLICTING OPINIONS.

There must have been almost immediate and universal unanimity as to the symptoms, gross appearance, and histology of papilloma of the larynx, since there has been little variation in discussions of these matters since the time of the earliest published reports; but at that point agreement ceased. Statements as to etiology have ranged from a firm belief in either chronic irritation,[8] chronic inflammation,[9] nasal obstruction,[10] or a constitutional diasthesis,[11] to a candid admission that the cause is yet to be determined; nor is the latter belief the most ancient.[12] Between these extremes, enough case reports have linked a specific trauma[13] or a particular infection[14] with the subsequent development of papilloma to further cloud the picture of etiology.

In like manner, authorities have differed greatly on the sex preference and the age incidence of this disease. Frequently it has been stated that these growths are more common in males;[15] but at least one observer found just the opposite to be true in his series of cases,[16] while reports from other sources indicate little, if any, sex preference.[17] Many authorities consider the disease primarily one of infancy and early childhood;[18] others believe it to be chiefly associated with middle age;[19] and yet a third group presents evidence that the symptoms of laryngeal papillomata may appear at any age, from birth to death.[20]

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[7] Early writers, such as Bosworth and Shurly, agree that Mackenzie was first to perform this service.

[8] Ehrmann and Horace Greene: cited by Mackenzie, op. cit., pp. 8-9.

[9] Mackenzie, ibid.; also Equen.; Ann. Otol., Rhinol., Laryngol., 50:776-782, Sept., 1941.

[10] Lennox Browne: The Throat and Nose and Their Diseases, 5th ed., pp. 650-657. Also, Ingersoll; reprint from The Laryngoscope, Aug., 1899.

[11] Brose: J.A.M.A. (reprint), 44:874, 1905.

[12] See Babcock: Surg. Clin. North America, 14:301-306, 1934; New and Erich, loc. cit., 1934; Ferguson and Scott, New Eng. Jour. Med. 230:477-482, April 20, 1944; Green, Med. Jour. Australia, 1:627-628, May 2, 1953; Rubin, Canad. Med. Assoc. Jour., 71:572-575, Dec., 1954.

[13] See El Mofti: Laryngeal Polpoid Growths Following Endotracheal Anesthesia, Jour. Laryngol. and Otol., 63:759-761, Dec., 1948. He reports eight cases from the literature and adds one.

[14] Ballinger, in Diseases of the Nose, Throat, and Ear, p. 542, noted measles to be "apparently a prolific exciting cause" of laryngeal papilloma.

[15] Opinions expressed by Ballinger, Babcock, and Rubin, in the respective works or places previously cited.

[16] Ferguson: loc. cit.

[17] New and Erich: loc. cit.; Jackson: Trans. Am. Acad. Ophtholmol. (reprint), 1921; Holinger et al.: Ann. Otol., Rhinol. and Laryngol., 59:547-564, June, 1950.

Statements about the mortality rate vary widely, too. The oft-quoted phrase, "always benign and usually self-limited," is liable to misinterpretation if taken out of context. Jackson and Jackson, probably first to use the expression,[21] hasten to add that neglect to remove the growths from the throat at proper intervals will endanger a child's life by reason of asphyxia. Ferguson and Scott use almost the same words quoted above, but go on to say that the condition is clinically treacherous, and cite fatalities. Bosworth, writing in 1892, did not consider the presence of "benign tumors of the larynx" any menace to life;[22a] Ward, writing more than half a century later, implied agreement with respect to adults only when he asserted that in the adult the growth, if completely removed does not recur and does not spread;[22b] however, this view does not seem to be widely shared at the present time. Diametrically opposed to such sanguinity is the writer who believes that in childhood papilloma of the larynx usually has a fatal outcome,[23] while others assert that in children under five years the mortality rate probably exceeds that of cancer of the larynx in the adult population.[24] Another writer reports two intractable cases in adults,[25] and still others boldly assert that papillomata may recur repeatedly after removal, at any age.[26]

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[18] Green loc cit, says symptoms are most commonly noted about age two, Bradburn The Laryngoscope 51 1105-1113, Nov 1951 says symptoms usually appear before age 5 See also opinion of Casuit quoted by Roy, Ann Otol Rhinol and Laryngol 11 482-490 reprint 1902

[19] Ingersoll The Laryngoscope reprint August 1898 also New and Erich loc cit and Tebold quoted by Roy, loc cit

[20] Jackson and Jackson Diseases and Injuries of the Larynx on p 361 report cases, age newborn to 84 years Bosworth A Treatise on Diseases of the Nose and Throat Vol II pp 718-720 refers to several congenital cases

[21] Op cit p 362

[22a] Op cit, p 716

[22b] Ward and Hendrick Diagnosis and Treatment of Tumors of Head and Neck, p 596

[23] Zalin Jour Laryngol Otol 62 621-626 Oct 1948 citing Holding 1929

[24] Ferguson loc cit and Zalin loc cit both citing Crowe and Breitstein, 1922

[25] Cohen South Med Jour 26 621 625 July, 1933

[26] Equen loc cit also Amarante A M A Arch Otolaryngol 58 94-95 July 1953

As to the possibilities of malignant degeneration, we again find a disquieting lack of agreement. The *tendency* of papilloma of the larynx toward malignant change is variously said to be:

1. Non-existent in children;[27]
2. Rare at any age; probably only co-incidental;[28]
3. Present at all ages;[29]
4. Potential, in adults only;[30]
5. Always actively present in adults; the multiple type is a pre-cancerous lesion.[31]

This summation at least gives one a wide range of choice.

In view of the foregoing recital of differences in opinion, it is no surprise to find that methods of treatment have run the gamut from a "hands off" policy which admits the necessity for tracheotomy only in the event of acute respiratory distress, through a variety of local and systemic medications, to simple excision, radical surgery, X-radiation, radium, diathermy, or many and varied combinations of these procedures.

A normal shift in opinion and method of treatment, to conform with progress in science, fails to explain these strikingly antithetical conclusions, outlined above, for many of them have marched side-by-side, chronologically speaking. In studying the literature on this topic we no sooner feel that a generality has been established than we find report of an outstanding exception. How can we reconcile such divergencies? What is there in the character of this disease to explain its variety of seemingly contradictory forms of behavior? We are reminded of the ancient fable of the blind men and the elephant; the thought arises that perhaps our failure to understand the

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27. Willis: Pathology of Tumors, p. 302, 1948; also Ferguson, loc. cit., and Zalin, loc. cit.

28. Babcock: loc. cit.

29. Cohen: loc. cit.; Figi: Minn. Med., 21:553-558, Aug., 1938; Ewing: Neoplastic Diseases, pp. 922-925.

30. Willis: op. cit., p. 302; Putney: A. M. A. Arch. 385, April, 1955.

31. Harris and Wattleworth: A. M. 1- June, 1951.

essential nature of laryngeal papilloma may explain the conflicts found in the various accounts of the disease

This paper will review the theories of etiology and the principal methods of treatment of laryngeal papillomata and attempt to evacluate antibiotic therapy of the disease, with particular reference to aureomycin

DEFINITION

Stedman's Medical Dictionary, 18th edition (1953), defines papilloma as "A circumscribed overgrowth or hypertrophy of the papillae of a cutaneous or mucous surface, the papillomata include warts, condylomata, cutaneous horns, and various polypi"

It is generally agreed that laryngeal papilloma are benign papillary masses springing from a small base, may be single or multiple, and may occur in any part of the larynx [32]

HISTOLOGY.

It is also generally agreed that papilloma arise from the squamous epithelium, that their cells are well differentiated, that they do not invade the stroma or extend below the basement membrane, and that their core consists of a rather vascular connective tissue Lipschutz sometimes found inclusion bodies in the epithelial cells [33]

Many investigators point out that while papillomata in children and in adults have identical characteristics, microscopically, yet their clinical behavior differs,[34] but as to the nature of that difference there is not complete accord Broyles asserts that in adults the growths occur singly, and that while their complete removal results in a cure, the unoperated papilloma may become malignant Amarante feels that, in the adult, papilloma is unpredictable, it may be cured by a single removal, or may recur repeatedly Ward is convinced that in adults the completely removed papilloma does not recur and does not spread Amarante expects recurrence during childhood, but recommends removal only to facilitate respiration. Broyles expects multiple growths in children and implantation on adjacent sites; he does not find extirpation satisfactory in children. Both Amarante and Broyles anticipate disappearance of the growths at puberty, and in this Ward concurs.

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[32] Babcock loc cit

[33] Cited by Ferguson loc cit

[34] Broyles Bull Johns Hopkins Hosp 66 319 322 May 1940 also Boles The Laryngoscope 53 101 111 Feb 1943 Ward and Hedrick op cit pp 596 597 1950

GROSS APPEARANCE.

Seen through the laryngoscope, papillomata are characteristically mulberry-like masses with a nodular surface. They vary in size from the tiny, asymptomatic "millet seed" which is occasionally discovered incidentally or at autopsy, to fungating masses larger than an olive. They may project from the mucosa of the cords, the anterior commissure, or anywhere else; the anterior third of a vocal cord is a common site for these growths.

In color they range from grayish-pink or yellowish-pink to an angry red when engorged. In the latter state they have been thought to be less amenable to treatment.[35]

SYMPTOMS.

Symptoms of papilloma result from alterations of the laryngeal function which cause changes in respiration and phonation. Usually phonation is first affected; there is an alteration in the quality of the voice, followed by a hoarseness which progresses to complete aphonia. In the meantime the respiradeath from asphyxia during a choking fit at the dinner table, without any prior manifestation.[36] In other instances, laryngeal papillomata have been first revealed at autopsy, after death from totally different causes.

Repeated upper respiratory infections, so frequently associated with laryngeal papilloma, are a characteristic result of interference with normal function. The child who has a series of "colds," and also demonstrates either hoarseness or an alteration in phonation, should be suspected of papilloma.

DIAGNOSIS.

Diagnosis is made by both indirect and direct laryngoscopy, and confirmed by histological examination of the removed tumor mass. In differential diagnosis the age of the patient helps decide which alternative diagnoses must be ruled out. In the adult one must consider the possibility of carcinoma, leukoplakia, hyperkeratosis, tuberculosis, and syphilis. With children one must think of congenital anomalies, such as webs and cysts, and of laryngeal stridor. Holinger states that polyps, vocal nodules, "screamer's nodes," and chronic laryngitis are common to both age groups, and these conditions, too, must be ruled out.

ETIOLOGY.

Chronic Irritation.

Probably the earliest theory of etiology which still has its adherents is that expressed by Bosworth in 1892,[37] and by Browne even earlier.[38] They believed that papilloma of the larynx is the result of chronic irritation. Browne traced more than half of a series of 26 cases to over-use of the voice. More convincingly, in a series of 300 cases Fauvel[39] found the greatest incidence among those whose work required special or excessive use of the voice, such as ministers.

While apparently accepting this theory of etiology, Bosworth also pointed out that not even a normal amount of functional activity is necessary to produce the growth, and he cited Mackenzie's case of papilloma in the larynx of a deaf-mute.

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[36] Ibid.

[37] Op. cit., pp 717-718.

[38] Cited by Bosworth, op. cit., p. 717.

[39] Ibid.

Later Lennox Browne voiced the opinion that chronic nasal obstruction is an important "pre-disposing local cause" of papilloma.[40] With this Ingersoll agreed, as did Ballinger, some years later.[41]

Chronic Infection.

Shurly presented another theory.[42] He expressed the then current opinion of Mackenzie, Cohen, Semon, and Sajou by indicting "chronic laryngeal catarrh" as the "principal etiological factor" in producing papilloma. These men pointed to the almost constant respiratory infections present in many cases of papilloma, and interpreted that infection as the most probable cause of the growth, whereas more recent writers have concluded that such infection is more probably the *result* of the impaired respiratory drainage induced by the obstructing growths.[43]

The much earlier suspicion that lues is the antecedent of papilloma was discounted at the beginning of the Twentieth century. Mackenzie[44] went so far as to assert that both syphilis and tuberculosis "exercised an antagonistic influence on the development of new growths," including papillomas of the larynx, but Ballinger reasoned that any systemic infection would lower body resistance and thereby be more likely to pave the way for the development of papillomas.

Ballinger seems to have favored the theory of an infectious origin of the disease, specifying measles as an "exciting cause." Whatever influence he attributed to mouth breathing, or to other "local irritants," seems to have been on the basis of such an irritant preparing the soil for a more favorable growth of the responsible infection.

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[40] Op. cit., pp. 650-657.

[41] In 1911.

[42] A Treatise on Diseases of the Nose and Throat, 2nd ed., pp. 564-565, 1905.

[43] Rubin: loc. cit.

[44] Cited by Ballinger, op. cit., p. 542.

Virus.

More recently we have observed the rise of a new theory: *the hypothesis that a viral agent is the chief cause of laryngeal* papilloma has been repeatedly tested and seems to be gaining acceptance.

Diseases now known to be caused by a virus were recognized hundreds of years ago, but their etiology was not suspected until a Russian scientist stumbled on a part of the mystery while studying the plant disease called "tobacco mosaic," in 1892.[45] He found that the infectious agent was in the juice of the plant,[46] and was filterable; but he failed to recognize the significance of his experiments, and it remained for the Dutch scientist, Beijernick, to realize that a new disease-producing agent, different from bacteria, had been discovered.

During the past decade, viruses have been subjected to much careful research. Many earlier concepts are now being discarded. while others are under scrutiny; but certain outstanding characteristics of viruses appear to be verified.

From our point of view, the most important of these are:[47]

1. Viruses are able to pass through filters which hold back the smallest known bacteria.

2. Viruses are parasites at the cellular level.

3. Viruses are able to multiply with astonishing rapidity, but only within the living cells of certain susceptible hosts.

The most generally accepted criterion of a virus is that it can be transmitted by a cell-free filtrate.[48]

The first recognized virus disease of animals was foot and mouth disease. Soon it was proved that a virus causes certain types of tumorous growths in fowl, and later this same agent was demonstrated to cause some forms of growths in animals as well.

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[45] Stanley Recent Advances in the Study of Viruses, Science in Progress, ed by Rivers, First Series, pp. 78-111.

[46] Cf observations of Hubbard, Boies, and Kirchner, referred to in this ms, p 15

[47] Burrows et al.: Textbook of Microbiology 16th ed, pp. 713-735. Frobisher: Fundamentals of Microbiology, 5th ed, pp 61-64 Stanley: loc. cit, pp. 80-83. Greenstein and Haddow: Advances in Cancer Research, Vol II, 1954, p 405

[48] Burrows' op cit, p 715.

As early as 1891 it was observed that contact with the cut surface of a common wart on the hand produced similar lesions on the contacting skin.[49] Experiments made by Variot in 1893, and by Judassohn in 1894, supported the hypothesis that some agent in the blood from verruca was capable of transmitting the growth from one host to another. Encouraged by these observations, Udo J. Wile made a series of experiments from which he concluded that warts can be produced by a virus which will pass through a Berkefeld filter.[50]

It had long been suspected that papilloma of the larynx, genital warts, and common verruca are all manifestations of the same pathological phenomenon, for as early as 1853 Buck had noted their similarity.[51] This suspicion was given strong support in 1923 when Ullman successfully innoculated the vaginal membrane of a dog with laryngeal papilloma, using bacteria-free filtrate.[52] His experiment simultaneously presented convincing evidence that genital warts and laryngeal papilloma are of the same etiology, and that a virus is responsible for both.

In the meantime, Hubbard[53] noted evidence of contact reproduction in "warts, bladder polyps, and laryngeal papillomas." He pleaded for extreme care and gentleness in the handling of these growths warning strongly that "the very juices of the neoplasm may be the agency of transplantation."

Boies,[54] favoring the viral theory, added, "It has been my clinical impression that the rapid appearance of papilloma in the trachea of a small child after scalping off multiple laryngeal growths was probably the result of expressing secretion from these growths in my efforts at removal."

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[49] Wile and Kingery: J. A. M. A., 73:970-972, Sept. 27. 1919.

[50] Ibid. Also, Warren: Warts; Verrucae, Viral and Rickettsial Infections of Man, 2nd ed., ed. by Rivers, p. 669.

[51] Trans. Amer. Med. Assoc. (reprint), 6:510-535, 1853.

[52] Boies: loc. cit.

[53] Hubbard and Galbraith: Arch. Otolaryngol., 1:70-78, Jan., 1925.

[54] Writing in 1943.

Eight years later Kirchner[55] voiced a similar thought, saying that trauma, "even sponging with gauze," seemed to spread the growth. He observed that the majority of extensions were found along the line of previous instrumentation.

Ferguson, reporting on his series of cases in the following year, admitted that a filterable virus might be a cause of papilloma but felt that not enough evidence had yet been presented to prove the point beyond dispute.

Zalin, writing a few years later,[56] accepted the virus theory and advanced the hypothesis of antibody immunity to account for spontaneous remission before puberty. With respect to that regression of the growths which has so often been observed at puberty, he suggested, "it is highly possible that the mature epithelium presents a barrier to virus activity not present in the immature tissue."[57]

As more became known about the behavior of viruses, the theory of viral etiology gained adherents. Holinger accepted it, modified by a recognition of the probable influence of hormones, much in the manner expressed by Zalin, and based a new therapeutic regimen upon that assumption.[58]

By 1952, Joel Warren was able to assert: "It has been definitely shown that an agent filterable through Berkefeld candles of all grades of porosity is responsible for warts. . . . Furthermore, most workers believe that a single etiological agent is responsible for the different types of warts mentioned."[59] In his list of warts, Warren specifically includes laryngeal papilloma; we are reminded of that ancient description, "warts in the throat."[60]

Green agreed with Holinger as to etiology, and reported a case[61] treated successfully by Holinger's method. By 1954 Rubin[62] felt justified in stating that the viral theory of etiology is now favored by the majority of authors.

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[55] Laryngoscope 61 1022-1029, Oct, 1951

[56] In 1948

[57] See discussion under Medical Treatment, this ms, p 25

[58] His theories and methods are discussed in this ms, p 28

[59] Warts Verruca, in Viral and Rickettsial Infections of Man edited by Thomas M Rivers 2nd ed p 669

[60] This ms, p 1

[61] Loc cit, 1953

[62] Loc cit

FORMS OF TREATMENT.

Before the invention of the laryngoscope, any recognizable papilloma probably was of considerable size and required heroic measures. Removal usually was accomplished through an incision made in the neck, though Bosworth reports that at least four workers had successfully removed laryngeal papilloma through the mouth, prior to 1892. More commonly, tracheotomy alone was done, and that only when respiration became greatly embarrassed. There were so many early instances of spontaneous regression of the growth that Ballinger wrote, in 1911, "Like warts on the skin, papillomata of the larynx come and go without any apparent reason."[63] This characteristic of the disease may explain why so many different forms of treatment have been inaugurated, used successfully by one or two men, and then tried without benefit by later workers. It also makes the evaluation of any form of treatment extraordinarily difficult.

Since various combinations of methods have been used, we shall consider treatment under the headings of Surgical, Physical, and Medical, according to the type which predominates in a given regimen of therapy.

Surgical.

From ancient times, when purely mechanical limitations may have dictated the method of treatment, tracheotomy has been a favorite procedure, not only as an emergency measure but also as a primary treatment for laryngeal papilloma. Roy early (1902) expressed a conviction that voice rest, obtained by tracheotomy, provided the best possible therapy, particularly in children. Brose, as well as other of Roy's contemporaries, agreed with that view, as do such modern writers as Zalin (1948), Ward (1950), Gorrell,[64] and Amarante (1953).

On the other hand, New,[65] McKinney,[66] and Ferguson (1944) saw no benefit in tracheotomy save as a temporary emergency measure. The hazards of dependence on tracheotomy as a routine therapy have been pointed out. *First,* sole reliance on tracheotomy may mask extensions of the disease which are incompatible with life, as in the case reported by Kirchner.[67] *Second,* unless the patient is closely and continuously supervised by a competent person, the tracheotomy cannula may become clogged or dislodged, or asphyxia may result from clumsy attempts to change the cannula at home. Deaths from such causes have been reported by Hitz,[68] Gorrell,[69] Buck,[70] and others. *Third,* malignant degeneration may take place undetected, as in the two fatal cases reported by Walsh.[71] One child wore a tracheotomy tube continuously from the age of 34 months until death from unsuspected malignancy at the age of 12; the other patient had continuous tracheotomy from age 3 until death from malignancy at age 11.

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[63] Op. cit., p. 545.

[64] Canad. Med. Assoc. Jour., 67:425-427, Nov., 1952.

[65] Ann. Otol. Rhinol. and Laryngol., 30:631-636, Sept., 1921. He mentions a woman age 22, whose larynx was filled with papilloma and who had worn a tracheotomy tube since age 2.

[66] Memphis Med. Monthly., reprint, Sept., 1915.

Forceps removal, repeated as often as necessary, probably has more adherents than any other method of treatment. Brose[72] recommended endolaryngeal removal in conjunction with tracheotomy. Jackson[73] reminded us that the disease is "self-limiting" in children, and predicted recurrences until the "time limit" is reached, no matter what form of treatment is employed; therefore, he recommended scalping off the projecting parts as often as is necessary to maintain an adequate airway, enjoining care that the normal tissues be not injured. Cohen[74] felt that surgical extirpation is the best method for adults as well as for children, asserting that a single procedure often suffices, but that two or more sittings may be required if the growth be multiple. Babcock[75] agreed. Broyles[76] felt that endoscopic removal is best at all times for adults, but effective for children only if accomplished during a period of either quiescence or regression, as determined by observation. Equen[77] preferred removal "flush with the surface" for children. Ferguson[78] thoroughly agreed with Jackson.

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[67] loc cit

[68] Am Jour Path (reprint), 8 333-338 1938

[69] loc cit 1952

[70] loc cit, 1853

[71] Laryngoscope, 60.1110-1124, Nov, 1950

[72] Paper published in 1905, the following dates are given to show continuity· 73 Paper published in 1921; 74 Paper published in 1933, 75 Paper published in 1934, 76 Paper published in 1941 77 Paper published in 1941, 78 Paper published in 1944, 79 Paper published in 1951; 80 Paper published in 1954

Harris[79] reviewed 116 cases, some treated by surgery and some by X-ray, and concluded that in the groups under his observation, surgery had given the better result. Rubin[80] stated that the treatment of choice, at any age, is forceps removal, repeated as often as necessary. The most recent opinion we have seen, that of Putney, published in 1955, expressed strong preference for surgical removal alone, mainly because such procedure permits repeated examination by the pathologist throughout the course of the disease, which this author regards as always potentially malignant.

To present just one opinion on the opposite side, we note that Hollingsworth[81] objected a few years ago that surgical removal requires "long hospitalization and repeated instrumentation," and advocated instead a form of medical treatment, which will be discussed later.

Physical Treatment.

In 1921, New reported on a series of 26 children seen at Mayo's over a period of six years. These patients, many of whom had repeated surgical excisions and/or tracheotomies previously, were treated with radium, under general anesthetic, using the Lynch suspension apparatus. These treatments, given at intervals of six to eight weeks, varied in number from one to six, and were sometimes supplemented with external radium. The patients ranged in age from 10 months to 12 years. He discounted nine cases for various reasons,[82] found improvement in six who still required further treatment, and reported a clear larynx in 11 of the group. The exact length of follow-up at the time of the report is not specified. This author found none of the previously deplored complications attendant upon radiation therapy; however, it is interesting to note that in a paper presented some years later, in 1938, he expressed a preference for diathermy.

Foster,[83] while insisting upon the necessity for tracheotomy in every case, recommended careful X-radiation as an adjunct, specifying that the "improved methods" of X-ray be used. His report was concerned primarily with children, but about the same time Cohen also expressed approval of X-ray after surgery for adults, if done with great care, though he preferred excision alone for both age groups.

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[81] Hollingsworth et al.: Arch. Otolaryngol., 52:82-87, July, 1950.

[82] One died after emergency tracheotomy, one died at home for want of tracheotomy, four did not complete the course of treatment, two were "unable to return," and one was lost to follow-up.

[83] South. Med. Jour., 26:625-630, July, 1933.

About ten years later, Salinger[84] condemned irradiation as "dangerous to children and valueless to adults;" he reported arrested development of the larynx of a child following irradiation. He considered such a result an inherent danger of irradiation, and concluded that the procedure had already been generally discarded.

Diathermy was advocated by Hubbard as early as 1925, and 13 years later by New and Erich. These men felt that "the juices of the neoplasm" were a means of its spread, and hence preferred a bloodless method of treatment. Their reasoning was based on the same observations which later led to annunciation of the viral theory of etiology.[85]

Medical Treatment.

Attempts at medical methods of conquering laryngeal papilloma began at a fairly early date. Delevan,[86] Roy,[87] Kyle,[88] and Stuckey[89] independently reported success in the treatment of adult cases from local applications of alcohol, usually in conjunction with tracheotomy; but others used this method without benefit. Though approving alcohol, Roy expressed belief that the voice rest provided by the concomitant tracheotomy was the better part of the therapy. This author also noted spontaneous cure in a 9-year-old girl after surgery was unavoidably delayed; her only treatment was repeated swabbings with cocaine, to which he attributed no curative power.

Shurly[90] is reported to have used Thuja Occidentalis with good results; Ballinger recommended arsenic as the best of internal remedies, but gave no details. Katz[91] advised local applications of magnesium oxide after excision, to discourage regrowth. A variety of other local medications are mentioned in the literature, but none seems to have gained a wide or lasting acceptance. The search for a more effective treatment continued.

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[84] Ann. Otol., Rhinol. and Laryngol., 51:273-277, March, 1942.

[85] See this ms., p. 15 ff.

[86] Cited by Ballinger, op. cit., p. 544; strength not given.

[87] loc. cit., 1902; daily applications, strength not specified, were followed by regression of growths; no details.

[88] A Textbook of Diseases of the Nose and Throat, 4th ed., 1907, pp. 221-224. He reported "injections of pure alcohol into these benign growths are highly beneficial."

[89] Tr. Am. Laryngol., Rhinol. and Otol. Society (reprint), 1913. He recommended alcohol of increasing strength daily, indefinitely.

[90] Cited by Ballinger, op. cit., p. 544.

In the meantime, the observation that the papilloma of childhood tended to disappear at puberty gave rise to a theory of hormonal relationship, and pointed the way to experiments with estrogens to ameliorate and/or reverse the course of the disease in children.

Te Linde and Brawner[92] had demonstrated that the vaginal mucosa of young girls could be quickly altered from the simple, infantile type to the stratified adult type of squamous epithelium, thereby making it more resistant to infection.

The question then arose, since papilloma of the larynx tends to disappear at puberty, would a forced change to adult type epithelium hasten this disappearance? Turning to the hormones, Holt[93] experimented unsuccessfully with testosterone; in his subjects, secondary sex characteristics immediately appeared, but the papillomata were not affected.

Broyles then began treatment of five patients with estrogen, resorting to surgical removal of the growths as necessary. His patients in this series were 5 to 7 years old, and all had been previously treated elsewhere, usually by repeated surgical removal. In each instance, Broyles[94] was able to report a normal voice and a clear larynx within periods ranging from two months to one year after the first of a weekly series of applications of Amniotin. Four of these patients had been followed for approximately one year at the time of his report, and no return of symptoms had been observed; but in 1944, Ferguson reported a trial of Amniotin on a single case with no success. It is not known whether or not the dosage and duration of treatment were identical with those employed by Broyles.

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[91] Cited by New and Erich, 1938.

[92] Am. Jour. Obst. and Gynec., 30:512-523, Oct., 1935.

[93] Cited by Broyles in 1940.

[94] Paper published in 1940.

In 1948, Zalin reported treatment of three patients with Organon, supplemented in one instance with Stilbestrol. He felt that this method was successful, in that the lumen was cleared by the treatment, and in two cases remained clear for the three-year period up to the time of his report; the third case was still under treatment.

Ward, in 1950, reported that local applications of Amniotin seemed to hasten the change from infantile to adult type epithelium, and that once such a change was accomplished the growth could be completely excised without fear of recurrence. In this there is an echo of the earlier observation that in children the surgical extirpation of papillomata is not successful unless the growths have reached a stage of quiescence or regression.

Opposed to those who found hormonal therapy effective, Hollingsworth[95] noted that Amniotin had been used without relief in one of the cases which came to his attention, and as recently as 1953, Amarante regarded hormonal treatment as wholly unsuccessful.

In the meantime, Hollingsworth and associates had begun to study the problem from the viewpoint of comparative histology. They remarked upon a previously-discovered similarity between venereal papilloma (verruca acuminata) and laryngeal papilloma, both in histologic appearance and in tendency to recur.[96] They appear to have disregarded the earlier work of Ullman and of Wile, though the conclusions of both tended to support Hollingsworth's theory. Noting that venereal papillomata had been treated successfully with resin of podophyllum,[97] they began experimenting in the treatment of laryngeal papilloma with the same preparation, which is said to have the property of arresting mitosis. These workers also observed that it immediately caused neoplastic tissue to turn gray in color.

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[95] Paper published in 1950

[96] Eggleston and Wolff Histopathology of the Ear, Nose, and Throat. 1947, cited by Hollingsworth

[97] I W Kaplan: Condylomata Acuminata: New Orleans Med and Surg Jour., 91 388-390, Feb., 1942, cited by Hollingsworth and not included in the appended bibliography.

Reporting on a series of five cases, each of which had been subjected to various other prior treatments[98] without improvement, Hollingsworth concluded that repeated weekly topical applications of a 10 to 15 per cent solution of podophyllum in 95 per cent alcohol, over a period of several weeks, resulted in regression, if not total disappearance, of the papilloma. After the first three or four treatments he lengthened the interval between treatments, as the situation indicated. He also removed the growths surgically as often as seemed necessary and found that the continued use of podophyllum progressively facilitated subsequent removals. It is interesting to note his comment on the use of antibiotics: "prolonged courses of treatment with penicillin, streptomycin, and Aureomycin apparently have no effect in inhibiting the growth of laryngeal papilloma."

Hollingsworth's criticism serves as a back-handed introduction to the most recent form of medical treatment, the antibiotics.

The use of antibiotics in treatment of laryngeal papilloma was the inevitable result of increasing interest in the viral theory of etiology. When the specificity of Aureomycin for certain virus infections had been demonstrated, Holinger[99] was probably first to report its successful application to papilloma of the larynx. In 1950 he reviewed a series of seven cases which he had found amenable to Aureomycin. This he had administered in dosages of 25 to 50 mm. per kilogram of body weight, depending upon the individual condition, over periods of from two to four weeks, following excision; in three instances he gave a second course of Aureomycin over a three-week period. The results were encouraging, and he remarked that the larynx appeared smoother than when surgery alone was used. Because of the small number involved and the comparatively brief span of time which had elapsed since treatment, he felt that it was too early to draw firm conclusions at the time of his first report. A year later he was able to add two more cases to his list of those successfully treated and to bring the previously-treated cases up to date.[100] In three of the first group he had obtained a clear larynx, and there was obvious regression of the growths in the remaining four.

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[98] Prior treatments in each of the five cases were: a. Surgical excision and tracheotomy; Amniotin spray, but strength, frequency, and duration of treatment not specified. b. Aureomycin, sulfadiazine, and penicillin for associated septicemia, given over 16-day period; unspecified dosage. c. Tracheotomy and Amniotin, unspecified dose; repeated courses of penicillin and streptomycin for recurrent respiratory infection; unspecified dosage; d. Surgical removal, twice. e. Surgical removal; repeated tracheotomy; radiation; hormonal therapy, no details.

[99] loc. cit., 1950.

A few followed Hollinger's lead at once. Green[101] soon reported a case in which use of Aureomycin brought about a dramatic disappearance of the papillomata in the larynx of a 3-year-old boy. The child had previously required many removals of the growth, and repeated tracheotomies. Finally he was given 200 milligrams of Aureomycin every six hours over a period of ten days. Regression of the growths was apparent within a few days, and at the end of three weeks his larynx appeared entirely free of papillomata on dirct laryngoscopy. He had been followed for two years at the time of the report, was in good health, with good voice, and a larynx which still appeared "perfectly clear."

McCart[102] reported the case of a boy, age 10, who suffered from dyspnea and required immediate tracheotomy. He had manifested symptoms of laryngeal papilloma for three years and had been treated with radium beams elsewhere. He was now given a course of Aureomycin,[103] which brought about relief of his symptoms and seeming regression in the growths; but within a few months he was readmitted with evidence of malignant degeneration, and soon died of massive pulmonary hemorrhage.

Shortly after Holinger's first report on his results from Aureomycin, Bradburn[104] published an account of the successful treatment of four patients with Terramycin. This group was comprised of three children and one adult, and had previously been treated elsewhere. After excision of the growths, Bradburn administered Terramycin, in doses of 50 mgm. per kilogram of body weight, daily for two months or more. In all four cases he found the response to be both immediate and gratifying, but at the time of his report, no case had yet been followed for more than six months.

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[100] Ann Otol, Rhinol and Laryngol, 60 496 (reprint), June, 1951.

[101] loc cit paper published in 1953

[102] Ann Otol, Rhinol and Laryngol, 63 498-499, June, 1954

[103] Dosage not specified

[104] loc cit, paper published in 1955

Opposed to these instances of successful treatment with antibiotics is Putney's conclusion that any improvement which has seemed to result from antibiotic therapy has in reality been due to the elimination of co-existing infection, and not to any successful attack upon the growth itself.

DISCUSSION.

Other observers have already commented that the continuing variety of treatments offered laryngeal papilloma indicates that the most effective method has not yet been found. It is amazing that so many workers have reported success with a given form of treatment, only to have later writers condemn it as unsound or ineffectual, a sequence of events many times repeated in the literature of this disease.

Possible explanations for this rather strange situation occur to the reader:

1. In some instances, the beginning of the treatment may have roughly coincided with the regressive phase of the disease, as suggested by Jackson in his paper of 1921.

2. While clinical manifestations and gross appearance of the growths are essentially the same, there may exist unsuspected differences in etiology which account for the variations in response to treatment. Even assuming that all laryngeal papillomata are of viral etiology, there is yet room for difference in the particular virus responsible in each of several different cases, particularly in different age groups, and probably even within the same age group. As investigation progresses, it becomes more and more apparent that the viruses offer an unexpectedly wide field for study and that their uninhibited behavior, their response to medication, and even their size is subject to infinite variety.

3. Over the years, and perhaps even at the present time, there have been some differences in pathological nomenclature. This is of especial importance in accounting for differences of opinion as to tendency toward malignant degeneration.

4. In view of the comparatively low and now declining incidence of the disease, no one worker has had opportunity of

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105. loc. cit., 1955.

observing response to a single therapeutic regimen in a large series of cases of any one age group. This has inevitably led to the formation of at least tentative conclusions on slighter evidence than is usually considered desirable; therefore we have of necessity approached the problem of treatment in a somewhat empirical manner.

Etiology.

The bulk of present evidence appears to favor the theory of viral etiology; however, we are reminded that this does not instantly provide a specific form of control, but rather opens the way to a new field of speculation and a comparatively unexplored method of treatment. In a sense we do not "know" nearly so much about viruses and their responses today as we did a few years ago, and perhaps our most important recent advance is a realization of this very lack of knowledge.

Incidence of the disease appears to be declining rapidly. Cunning postulates that improved nutrition is the principal cause, since the lower rate was apparent before common use of the antibiotics. If his hypothesis be true, it might be regarded as another bit of evidence to support the viral theory of etiology, since adequately nourished tissue would seem less liable to infection.

Varied Response to Antibiotic Therapy.

In reviewing the literature it appears that in instances where treatment with antibiotics has been followed by an obvious and continued regression of symptoms, or by complete disappearance of the papillomata, the dosage has been both massive and long-continued, by conventional standards. In the available reports of cases where the antibiotics have been found disappointing, the strength and extent of dosage has often not been specified, but we strongly suspect, from our own observation, that it has been uniformly inadequate.

In five cases which have come to our personal attention it appears that only massive doses have an inhibiting effect on the growths, and that lasting benefit results only if the medication is continued over a comparatively long period of time; a period of weeks, rather than hours, of months rather than days.

To illustrate, note that in Case I of the appended series of Case Reports, when conventional doses of antibiotic were given there was no appreciable change in the frequency with which the larynx refilled with growths; when the dosage was increased by 50 per cent the intervals between surgery were doubled; treatment with massive doses of Aureomycin over a period of weeks was followed by an immediate regression of symptoms and obviated the need for further surgery. The age of this patient, 53 years when first seen, discounts any possible hormonal influence.

In Case Report II, note that conventional doses of Duracillin in a 19-months-old boy had no appreciable effect on the papillomata, but massive doses of Aureomycin brought about almost immediate diminution of the growths.

In Case Report III, the patient's history included numerous attempts at removal, each of which resulted in regrowth. After one course of massive Aureomycin therapy there was a regression and an eventual disappearance of the papillomata. Thereafter the difficulty arose from the considerable amount of scar itssue which had accumulated over the years because of the repeated surgery, a result which argues well for the need to eliminate as many surgical procedures as can be omitted with safety, and to safeguard normal tissue as much as possible.

In Case Report IV, we find that it had been necessary to remove the growths at intervals of decreasing length until massive Duracillin therapy was begun. The next interval was three times the length of the greatest previous period between surgical procedures, but the growths still returned. Then massive Aureomycin therapy was instituted, and within 30 days the papillomata had definitely diminished in both number and size. The patient at that time was 3 years old. Aureomycin was continued and repeated laryngoscopic examinations verified the regression of the growths over a period of one year. At that time the cords appeared more smooth than is usually the case when only surgery is employed.

Case Report V shows much the same picture. When first seen, this 2-year-old child presented a glottis well filled with papillomata. After extirpation of the growths, massive doses of Duracillin were given for only three days. A diminution of symptoms resulted, so that the intervals between surgery were lengthened; but when massive Aureomycin therapy was begun, approximately a year after the patient was first seen, there was an almost immediate disappearance of symptoms, and after three months of Aureomycin, no papillomata could be seen on direct laryngoscopy.

Observations of only these five cases obviously cannot be offered as conclusive evidence of the efficacy of Aureomycin in the treatment of laryngeal papilloma; however, it will be remembered that in each of these cases an obvious regression of symptoms began only after massive doses of antibiotics, and that continued relief resulted only after massive doses of Aureomycin over a considerable length of time. In none of these cases could puberty have been a factor. Nor have other cases of the same age groups followed this same pattern of response, in our experience, when treated by either surgery alone or by surgery supplemented in any other manner.

A profile of 15 additional unpublished cases, treated at a University Hospital with varying amounts of antibiotics, is appended to demonstrate further the need for massive and prolonged medication, if these drugs are to be effective.

It is probable that in the past some of the discouraging reports on antibiotic therapy have resulted from judging its incidental effect, or rather lack of effect, on laryngeal papillomata when the medication was administered in the conventional amount, for the conventional length of time, in the specific treatment of an associated infection. Others have stemmed from an underestimate of the dosage necessary to control the disease while remaining within the limits of human tolerance.

CONCLUSIONS.

1. Present evidence indicates that laryngeal papilloma is of viral etiology.

2. Adequate dosages of antibiotics tend to control the disease.

3. It is the experience and observation of the writer that a sufficient dosage of Aureomycin, administered over a period of months, effectively supplements the surgical removal of stubborn papillomata and results in a clearer larynx than that usually achieved by means of surgery alone.

CASE REPORTS.

The following five cases, with diagnosis of laryngeal papilloma verified by histologic examination, have been under observation for five years or longer and have been treated with Aureomycin by either the writer or a colleague:

Case 1: C. H., a white woman, age 53 when first seen by a colleague in January of 1945. She complained of loss of voice, attributing her condition to the extraction of four teeth the preceding June, and asserting that she had experienced constant "throat trouble" since then.

First examination revealed greatly enlarged and seriously infected tonsils; accordingly, tonsillectomy was done. A very small papillomatous growth was observed on the left true cord, but was not removed at this time.

Five weeks later she returned to the office, again complaining of loss of voice. At this time, examination revealed growths on the left vocal cord extending from the anterior third up to the commissure. Biting forceps were used under direct laryngoscopy to remove all the extraneous tissue. A specimen sent to the laboratory was reported "fibro-epithelial papilloma with branching stalks of connective tissue covered with heavy layers of stratified squamous epithelium." Following this surgical procedure there was considerable improvement in the patient's voice.

Approximately eight months later she returned to the office, again unable to talk. She was readmitted to the hospital on Nov. 19, 1945, and this time a papilloma the size of a glass pin head was found at the junction of the anterior and the middle third of the left cord. This was removed with biting forceps, and the anterior two-thirds of each vocal cord, as well as the ventricular bands, were cleaned as well as possible with the forceps. The pathological report was again "benign papilloma." Improvement in voice again resulted, but only temporarily.

In May of 1946 she returned, again complaining of loss of voice. On May 21, multiple papillomata were removed, and again the voice improved.

In November of that year she again became unable to speak aloud. At that time, papillomata were found to cover the anterior two-thirds of the cords on both sides. The growths were removed by forceps on Nov. 22, and at this time considerable bleeding was encountered. Penicillin, 20,000 units every four hours, was given for three days, beginning Nov. 24.

She was re-hospitalized on May 12, 1947, with complete loss of voice. On this admission, growths were found on each vocal cord, the ventricular bands, and the anterior commissure. Small cup forceps were used to exenterate the papillomata from the entire glottis. Following this procedure, penicillin, 30,000 units every four hours, was begun May 16 and continued for one week.

The next hospital admission was March 9, 1948, again with total loss of voice Growths were removed from both cords on March 10, and then superficial coagulation was done Penicillin, 40,000 units every four hours, was given for one week

Following this treatment the usual improvement lasted more than a year, but on May 4, 1949, the patient was again admitted, with not only total loss of voice but dyspnea as well It was found that the vocal cords were again covered with papillomata, which were removed under direct laryngoscopy on May 5 Penicillin was repeated, as on the last admission.

This patient was not seen again until Oct 22, 1950, when papillomata were again removed, as before Penicillin was repeated, as previously

On Dec 12, 1951, the patient returned and presented a larynx fairly well filled with papillomata The following day the growths were again removed Aureomycin, 250 milligrams every six hours, was begun and continued for three weeks

Approximately a year later the patient was again hospitalized, but this time for posterior myocardial infarction She was able to speak only in a whisper, but the larynx appeared reasonably clear, despite the numerous surgical procedures She has not required further treatment for the papillomata, up to August of 1955

Case 2 H L G, a white boy, age 19 months when first seen by a colleague Dec 15, 1948 His parents reported continued hoarseness and difficult breathing since birth, he suffered from repeated upper respiratory infections There had been an exacerbation of symptoms following a recent acute bout of such infection Examination revealed multiple papillomata These were removed Dec 16, diagnosis confirmed by pathologic examination, and Duracillin was given, 1 cc daily for five days Immediate improvement was noted following surgery

He returned to the office in seven months, with hoarseness and dyspnea aggravated by recent upper respiratory infection On Aug 1, 1949, multiple papillomas were removed and Duracillin was given, as before

Because of dyspnea it again became necessary to remove the papillomata on Jan 12, 1950 At that time the larynx appeared to be completely filled with the growths Duracillin was given, as before

The symptoms recurred and the treatment was repeated seven times up to April of 1951 At that time the larynx was covered with papillomata, from the ventricular folds on down into the trachea and the air way was very poor Large amounts of papillomatous tissue were removed, piecemeal, from both sides thereby improving the airway Aureomycin therapy was then instituted, 100 milligrams every three hours for the first three days, and twice each day thereafter for a period of nine weeks During this period of treatment the larynx was twice inspected and showed progressive improvement No surgical intervention was necessary for eleven months

By May of 1952, dyspnea had returned and it became necessary to remove the papillomata No more Aureomycin was given

Surgical removal of the growths was necessary again in February of 1953

The child was last seen when the growths were removed again May 7, 1953 At that time, multiple papillomata were found, chiefly on the left side the superior surface of the left cord, the anterior two thirds of the left cord, and only the posterior surface of the right cord were involved This represented considerable improvement over some of the previous admissions, but indicated a trend toward another series of required surgical procedures of increasing frequency.

Case 3: R. J. O., a white boy, age 6 years when first seen by a colleague February, 1950. He was hospitalized Feb. 2, unable to speak above a whisper. He had a history of many previous hospitalizations for laryngeal papilloma, and a permanent tracheotomy since the age of 2. At the time of this admission he had little or no airway. Laryngoscopic examination revealed severe fibrosis and multiple papilloma. No surgery was attempted.

Three months later he was re-admitted and much of the growth was removed with bent cup forceps. He was then given a six-weeks' course of Aureomycin, 150 mgm. twice each day.

Seven months later, in December, he was re-admitted because of laryngeal stenosis. On examination he was found to be free of papilloma, but there was much scar tissue. The family was transferred elsewhere and the patient was not seen again.

Case 4: L. G. S., a white boy, age 2 when first seen, Oct. 21, 1948, complaining of "difficult breathing" for the past month. There had been a sudden onset of hoarseness in February, and this seemed to be increasing somewhat. Direct larygoscopy, done the following day, revealed multiple papilloma on the right cord, extending to the anterior commissure; diagnosis confirmed by biopsy. The growths were removed and the voice improved.

The child became hoarse and dyspnea returned. Papilloma were removed Feb. 27, 1949, and again on March 11, because of recurrent dyspnea. At this time both cords were covered.

Continued difficulty in breathing was relieved by a tracheotomy done March 29, 1949. He was closely followed and the tube was changed every four or five days at the office until late Summer of 1950.

On Aug. 22, 1950, multiple papillomata were removed under direct laryngoscopy, and Aureomycin therapy was begun.

*Aureomycin Spersoids, 4 cc. every three hours, given in milk, was con*tinued after the patient was dismissed from the hospital. The next examination, Sept. 19, revealed smaller and less numerous growths, a moderate improvement. The child was again closely followed, returning to the office for changes of the tracheotomy tube, as before.

By Jan. 23, 1951, laryngoscopy revealed considerable improvement. One papilloma was removed from the left cord, two from the right. Aureomycin therapy was continued.

On May 8, 1951, direct laryngoscopy showed only a small nodule on the posterior end of the right true cord. The left cord was thickened; both moved readily with respiration. On Sept. 18, 1951, a single residual papilloma was removed and the tracheotomy stoma was closed. The pathology report was essentially the same as the one made after the first removal of the growths. Aureomycin was now discontinued.

By Sept. 26, 1951, mirror examination revealed a good airway. The child had begun to talk in whispers. In another ten days he was talking very well. He was examined by mirror at frequent intervals, and by direct laryngoscopy on Feb. 29, 1952. At that time both true cords were smooth and the airway appeared good. No papilloma could be seen.

He has since been seen at intervals of approximately six months, the last office visit being May 23, 1955. At that time his voice was good; he had no hoarseness for the past three years. Mirror examination revealed both true cords to be pearly white, with free edges and superior surface entirely smooth. There was no evidence of recurrent papilloma.

This patient had been on Aureomycin continuously for 13 months He was 9 years old when last seen

Case 5· L C M, a white girl, age 22 months when first seen, Sept 3, 1948 She had no voice since an episode of measles, five months previously

Direct laryngoscopy was done Sept 9, 1948, and revealed a small growth on the left vocal cord This was removed on Nov 12, 1948, and proved to be papilloma The voice then returned

She was seen again on Feb 3, 1949, the voice was much better at that time, however, by May she had developed considerable hoarseness Soon thereafter she had pertussis, so that laryngeal treatment was postponed until Sept 16 At that time a direct laryngoscopy revealed a very poor air way and a paralyzed right vocal cord Tracheotomy was done the following day Thereafter she was kept under close supervision The papillomata returned, covering both vocal cords, and were removed, as well as possible on June 9, 1950, under direct laryngoscopy On Aug 28, 1950, we began the administration of Aureomycin Spersoids, 4 cc every three hours and on Sept 19, papillomata were again removed from the anterior third of both true cords.

The next examination showed marked improvement She was examined by mirror at regular intervals until Feb 6 1951, when direct laryngoscopy revealed a narrowed glottic chink but an adequate airway, without any evidence of papilloma In the meantime, the voice had improved and continued to do so

On April 17, 1951, direct laryngoscopy showed a small papilloma on the left cord This was promptly removed

Regular examination indicated progressive improvement, and on Sept 25 1951, the tracheal stoma was closed

On Feb 25, 1952, no papilloma could be seen, and the voice was good

By Jan 19, 1953, one small papilloma had appeared on the epiglottis, and there was appreciable hoarseness

On June 16, 1953, a papilloma was removed from the anterior portion of the right vocal cord and another from the posterior portion of the left cord Since that time repeated mirror examinations have failed to disclose any further return of papilloma Aureomycin was discontinued in April, 1954, after slightly more than three years of medication

When last seen, in February of 1955, the voice was strong, the true cords were pearly white and smooth, and breathing was normal

CASE PROFILES.

The cases reported in the following profiles were treated for laryngeal papilloma at the State University of Iowa Hospital, Department of Otolaryngology and Oral Surgery, and permission was given by that Department to abstract the necessary information from their records

In each instance, the diagnosis was confirmed by pathologist's report

AVO, FEMALE, AGE 4 YEARS. HOARSE PAST TWO YEARS.

Date Examined or Treated	Condition Found. Location of Growths	Surgical Procedures	Antibiotics Given	Dosage	Frequency	Duration	Other Treatment
2-2-49	Large papilloma on right true cord	Removed					
2-5-49	. . . .	Tracheotomy					
3-31-49	Right true cord	Removed					
6-25-49	Larynx clear	. . . .					
8-15-50	Cords covered with papilloma	Removed	Penicillin S-R	200,000 U.	2 × day	2 days	
3-7-51	Hoarse; right true cord	Removed					
4-27-51	Left true cord	Removed	Aureomycin	150 mgs.	4 × day	5 days	Podophyllum, 10%
6-19-51	No recurrence	. . . .					
9-21-51	Middle ⅓ both true cords	Removed	Aureomycin then	200 mgs. 50 mgs.	4 × day 4 × day	2 days 2 days	Diathermy to right Podophyllum, 10%, to both cords
11-16-51	No recurrence	. . . .					
1-2-52	Slight return, bilat.	Removed	Penicillin S-R Aureomycin	1 cc. 100 mgs.	2 × day Every 5 hrs.	2 days 2 days	
5-2-52	Ant. ⅓ left cord	Removed	Penicillin	400,000 U.	Daily	2 days	
4-9-53	No papilloma; granulation tissue on posterior *⅓ of left true cord*						

Time covered by this record: 4 years. Age when last seen: 6 years. Condition when last seen: considerably improved; no papilloma.

JH, FEMALE. AGE 3 YEARS. HOARSENESS AND DYSPNEA.

Date Examined or Treated	Condition Found. Location of Growths	Surgical Procedures	Antibiotics Given	Dosage	Frequency	Duration	Other Treatment
7-30-49	Ant. commissure and left true cord	Removed	Penicillin Crysticillin	25,000 U. 1 cc.	Every 3 hrs. Daily	2 days 2 days	Followed by
8-22-49	Right true cord, ant.	Removed					
10-29-49	Right true cord	Removed					
12-7-49	Left cord	Removed					
2-14-50	Right cord	Removed					
2-16-50	. . . .	. . . .	Penicillin S-R	1 cc.	Daily	1 week	
4-28-50	Left true cord	Removed					
6-19-50	Poor airway	Tracheotomy	Aureomycin	150 mgs.	2 X day	3 weeks	
6-28-50	Both cords	Removed					
10-3-50	Both cords	Removed	Aureomycin	125 mgs.	4 X day	5 days	
11-21-50	Both false cords	*Removed*	Aureomycin	125 mgs.	4 X *day*	10 days	
1-29-51	Many on left false cord; few on right, also on ant. commissure	Some removed	Aureomycin	125 mgs.	4 X day	15 days	
4-19-51	Greatly improved	. . . .	Aureomycin	150 mgs.	4 X day	21 days	
8-9-51	Improvement continues; voice good	Closed tracheotomy					
11-28-51	Few on right ventricle and ant. commissure	Removed	Aureomycin	125 mgs.	4 X day	12 days	
4-14-52	Small papilloma on both true cords	Removed					
1-20-53	Small papilloma on left true cord and ant. commissure	Removed	Penicillin	400,000 U.	Daily	7 days	
4-22-53	Clear	. . . .					
5-20-55	No recurrence; voice good	. . . .					

Time covered by this record: approximately six years. Age when *last seen*: 9 years. Condition when last seen: larynx clear; voice normal.

AVO, FEMALE, AGE 4 YEARS. HOARSE PAST TWO YEARS.

Date Examined or Treated	Condition Found, Location of Growths	Surgical Procedures	Antibiotics Given	Dosage	Frequency	Duration	Other Treatment
2-2-49	Large papilloma on right true cord	Removed					
2-5-49	. . . .	Tracheotomy					
3-31-49	Right true cord	Removed					
6-25-49	Larynx clear	. . . .					
8-15-50	Cords covered with papilloma	Removed	Penicillin S-R	200,000 U.	2 × day	2 days	
3-7-51	Hoarse; right true cord	Removed					
4-27-51	Left true cord	Removed	Aureomycin	150 mgs.	4 × day	5 days	Podophyllum, 10%
6-19-51	No recurrence	. . . .					
9-21-51	Middle ⅓ both true cords	Removed	Aureomycin then	200 mgs. 50 mgs.	4 × day 4 × day	2 days 2 days	Diathermy to right
11-16-51	No recurrence	. . . .					Podophyllum, 10%, to both cords
1-2-52	Slight return, bilat.	Removed	Penicillin S-R Aureomycin	1 cc. 100 mgs.	2 × day Every 5 hrs.	2 days 2 days	
5-2-52	Ant. ⅓ left cord	Removed	Penicillin	400,000 U.	Daily	2 days	
4-9-53	No papilloma; granulation tissue on posterior ⅓ of left true cord						

Time covered by this record: 4 years. Age when last seen: 6 years. Condition when last seen: considerably improved; no papilloma.

JH, FEMALE. AGE 3 YEARS. HOARSENESS AND DYSPNEA.

Date Examined or Treated	Condition Found. Location of Growths	Surgical Procedures	Antibiotics Given	Dosage	Frequency	Duration	Other Treatment
7-30-49	Ant. commissure and left true cord	Removed	Penicillin Crysticillin	25,000 U. 1 cc.	Every 3 hrs. Daily	2 days 2 days	Followed by
8-22-49	*Right true cord, ant.*	Removed					
10-29-49	Right true cord	Removed					
12-7-49	Left cord	Removed					
2-14-50	Right cord	Removed					
2-16-50	. . . .	. . . .	Penicillin S-R	1 cc.	Daily	1 week	
4-28-50	Left true cord	Removed					
6-19-50	Poor airway	Tracheotomy	Aureomycin	150 mgs.	2 × day	3 weeks	
6-28-50	Both cords	Removed					
10-3-50	Both cords	Removed	Aureomycin	125 mgs.	4 × day	5 days	
11-21-50	Both false cords	Removed	Aureomycin	125 mgs.	4 × day	10 days	
1-29-51	Many on left false cord; few on right, also on ant. commissure	Some removed	Aureomycin	125 mgs.	4 × day	15 days	
4-19-51	Greatly improved	. . . .	Aureomycin	150 mgs.	4 × day	21 days	
8-9-51	Improvement continues; voice good	Closed tracheotomy					
11-28-51	Few on right ventricle and ant. commissure	Removed	Aureomycin	125 mgs.	4 × day	12 days	
4-14-52	Small papilloma on both true cords	Removed					
1-20-53	Small papilloma on left true cord and ant. commissure	Removed	Penicillin	400,000 U.	Daily	7 days	
4-22-53	Clear	. . . .					
5-20-55	No recurrence; voice good	. . . .					

Time covered by this record: approximately six years. Age when last seen: 9 years. Condition when last seen: larynx clear; voice normal.

CJW, MALE. AGE 3 YEARS. WEAK VOICE SINCE BIRTH AND "CHOKING SPELLS" PAST TWO MONTHS.

Date Examined or Treated	Condition Found. Location of Growths	Surgical Procedures	Antibiotics Given	Dosage	Frequency	Duration	Other Treatment
8-13-49	Both *true* cords covered with papilloma	Removed					
9-2-49	Both true cords, but left worse	Removed from left					
9-21-49	Ant. ⅓ left cord	Removed					
3-17-50	*Multiple on both* false cords and on left true cord	Removed	Penicillin S-R	1 cc.	Daily	3 days	
4-10-50	Left true and false	Removed	Penicillin	400,000 U.	Daily	3 days	
8-21-50	No recurrence	. . . . .					
12-15-50	No recurrence	. . . . .					
2-16-51	No recurrence	. . . . .					
9-28-51	No recurrence	. . . . .					

Time covered by this record: approximately 2 years. Age when last seen: 5 years. Condition when last seen: larynx clear, airway normal, and voice satisfactory.

LL, FEMALE AGE 44 YEARS HOARSE SINCE CHILDHOOD

Date Examined or Treated	Condition Found Location of Growths	Surgical Procedures	Antibiotics Given	Dosage	Frequency	Duration	Other Treatment
7-20 50	Multiple, bilat.	Left cord stripped					
8 2 50	. . . .	. . . .	Penicillin	400,000 U	Daily	2 days	
8 21 50	Left cord worse than right	Removed from left	Penicillin	400,000 U.	Daily	2 days	
3-20 51	Ant ⅓ left cord, but smaller	Removed					
11-12 51	Small papilloma on ant left cord	Removed					
10 15 53	No papilloma	. . . .					
5-12 54	No papilloma	. . .					
3-21-55	No papilloma	. . . .					

Time covered by this record more than four years Age when last seen. 48 years Condition when last seen· larynx clear, voice improved

SKS, FEMALE. AGE 3 YEARS. HOARSE SINCE AGE OF 18 MONTHS.

Date Examined or Treated	Condition Found. Location of Growths	Surgical Procedures	Antibiotics Given	Dosage	Frequency	Duration	Other Treatment
9-29-49	Left true cord, posterior ⅔	Removed	Crysticillin	100,000 U.	Daily	5 days	
6-23-50	Left true and false	Removed	Aureomycin	250 mgs.	4 × day	3 days	
10-13-50	Both cords	Removed	Aureomycin	250 mgs.	4 × day	4 days	
12-28-50	Voice better	. . . .					
4-5-51	Larynx filled with papilloma	Removed	Aureomycin	250 mgs.	4 × day	3 days	
5-11-51	Right cord, post., and ant. commissure	Removed	Penicillin	400,000 U.	2 × day	2 days	
10-10-51	Moderate recurrence	Removed	Aureomycin	250 mgs.	4 × day	2 days	
11-29-51	Very small papilloma ant. ⅓ both true cords		Penicillin	400,000 U.	Daily	2 days	
5-6-52	. . . .	T & A					
8-12-52	Voice excellent Larynx clear	. . . .					
11-1-52	Larynx clear	. . . .					
5-5-53	Larynx clear	. . . .					
11-3-53	Larynx clear	. . . .					
5-8-54	Larynx clear	. . . .					
0-15-54	Larynx clear	. . . .					
-3-55	Larynx clear	. . . .					

Time covered by this record: approximately six years. Age when last seen: 9 years. Condition when last seen: xcellent.

GA, FEMALE. AGE 2 YEARS. DYSPNEA FOR THREE WEEKS.

Date Examined or Treated	Condition Found. Location of Growths	Surgical Procedures	Antibiotics Given	Dosage	Frequency	Duration	Other Treatment
1-21-50	Left cord covered	Tracheotomy	Penicillin	30,000 U.	8 × day	7 days	
1-29-50	. . . .	Removed					
2-8-50	Growths returned	Removed	Penicillin	25,000 U.	8 × day	1 day	
4-21-50	Cords, bilat., and ant. commissure	Removed	Penicillin	20,000 U.	8 × day	1 day	
5-4-50	Growths returned						Electric needle
6-9-50	Right true cord	Removed					
7-15-50	Right true and false cords	Removed	Aureomycin	250 mgs.	4 × day	2 months	
9-18-50	No papilloma	. . . .					
11-17-50	Left true cord	Removed					
1-21-51	Right arytenoid	Removed	No antibiotic because of history of rash after last med.				
6-15-51	No papilloma	. . . .					
8-22-51	No papilloma						
9-22-52	Ant. commissure, small papilloma	. . . .	Aureomycin *Spersoids*	250 mgs. in milk	4 × day	3 weeks	
11-30-53	No papilloma	. . . .					
7-28-55	Appeared normal						

Time covered by this record: Five and one-half years. Age when last seen: 7 years. Condition when last seen: larynx clear; breathing normal.

JHE, FEMALE. AGE 35 YEARS. HOARSE FOUR MONTHS.

Date Examined or Treated	Condition Found. Location of Growths	Surgical Procedures	Antibiotics Given	Dosage	Frequency	Duration	Other Treatment
3-31-50	Middle ⅓ right cord, ant. ⅓ left cord, and ant. commissure	Removed					
6-6-51	Slight return	Removed	Aureomycin	250 mgs.	4 × day	1 week	
7-12-51	Normal	. . . .					
10-22-51	Ant. ⅔ of both true cords	Removed					Podophyllum, 10%, one application
11-19-51	Ant. ⅓ left cord	Removed					
12-10-51	No recurrence	. . . .					

Time covered by this record: approximately 15 months. Age when last seen: 36 years. Condition when last seen: larynx clear and voice normal.

Note that there had already been longer intervals of freedom from symptoms, followed by recurrence, than that last recorded. This patient was lost to follow-up.

DB, MALE. AGE 6 YEARS. HOARSE SINCE AGE OF SIX MONTHS.

Date Examined or Treated	*Condition Found.* Location of Growths	Surgical Procedures	Antibiotics Given	Dosage	Frequency	Duration	Other Treatment
5-20-50	Both true cords and anterior commissure	Removed					
6-15-50	Growths returned	Removed	Aureomycin	250 mgs.	4 × day	3 days	
8-5-50	Growths returned	Removed					
12-11-50	Growths returned	Removed	Aureomycin	125 mgs.	4 × day	3 days	
3-26-51	Growths returned	Removed					
4-4-51	Growths returned	Removed	Aureomycin	250 mgs.	4 × day	5 days	
5-9-51	Growths returned	Removed	Aureomycin	250 mgs.	4 × day	5 days	
8-25-51	Airway very poor	Tracheotomy	Aureomycin	100 mgs.	6 × day	6 days	
8-31-51	*Improved*	. . . .	*Aureomycin*	125 mgs.	4 × day	22 days	
9-6-51	Continued improvement	. . . .	Aureomycin				
10-29-51	Status quo	Removed	Aureomycin	150 mgs.	4 × day	26 days	
2-7-52	Only slight return	Removed	Aureomycin	100 mgs.	4 × day	4 days	
3-14-52	Greatly improved	. . . .					
4-11-52	Continued improvement	. . . .					
5-20-52	Slight return	Removed	Aureomycin Spersoids	4 cc.	2 × day	4 days	
6-26-52	Left cord clear	. . . .					
8-12-52	No papilloma seen	. . . .					
9-30-52	Return on right side	Removed	Aureomycin Spersoids	2 cc.	3 × day	7 days	
12-5-52	No papilloma seen	. . . .					
1-10-53	Slight return	. . . .	Penicillin S-R	400,000 U.	Daily	4 days	
5-19-53	Ant. commissure and left false cord	Removed	Aureomycin Spersoids	4 cc.	2 × day	4 days	
11-8-51	Growths returned	Removed					
3-19-55	Growths returned	Removed					
7-18-55	Growths returned	Removed					

Time covered by this record: approximately five years. Age *when last seen:* 11 years. Condition when last seen: larynx badly scarred; growths recurrent.

JLP, FEMALE. AGE 2 YEARS. HOARSE SINCE AGE 6 MONTHS.

Date Examined or Treated	Condition Found. Location of Growths	Surgical Procedures	Antibiotics Given	Dosage	Frequency	Duration	Other Treatment
7-20-50	Both true cords and right false cord	Removed					
8-10-50	Severe dyspnea	Tracheotomy	Penicillin	200,000 U.	2 × day	6 days	
9-9-50	. . . .	Removed	Aureomycin	50 mgs.	3 × day	8 days	
10-6-50	Growths returned	Removed	Aureomycin	150 mgs.	2 × day	10 days	
11-14-50	One on right, few small ones on left	Removed	Aureomycin	150 mgs.	4 × day	4 days	
12-4-50	Much improved; few on right ant. true cord	. . . .	Aureomycin	150 mgs.	4 × day	4 days	
2-19-51	Returned, both true cords and right false	Removed					One application of podophyllum, 10%
4-21-51	Many on left, few on right	Removed					
5-17-51	Returned on right side	Removed					
7-20-51	Glottis almost closed	Removed, right					
7-24-51	. . . .	Removed, left					
9-21-51	Returned on both sides	Removed, right					
9-29-51	. . . .	Removed, left					
10-17-51	Returned, but fewer	. . . .	Chloromycetin	100 mgs.	4 × day	3 days	
11-10-51	Returned, few more	Removed	Penicillin	400,000 U.	Daily	2 days	
11-21-51	Returned, but fewer	Removed					
12-12-51	Returned, but fewer	. . . .	Penicillin	400,000 U.	Daily	2 days	
12-27-51	Further extension	. . . .	Penicillin	400,000 U.	Daily	2 days	
2-12-52	Status quo	Removed	Aureomycin	100 mgs.	4 × day	2 days	
3-12-52	Left true cord and interarytenoid fold	Removed	Aureomycin	50 mgs.	4 × day	2 days	

JLP, CONTINUED.

Date Examined or Treated	Condition Found. Location of Growths	Surgical Procedures	Antibiotics Given	Dosage	Frequency	Duration	Other Treatment
5-7-52	Both cords	Removed	Aureomycin	100 mgs.	4 × day	2 days	
5-28-52	Clearer; left false cord only	Removed	Aureomycin	100 mgs.	4 × day	2 days	
6-19-52	Few on both false cords	Removed					
7-25-52	Left side, true and false cords	Removed					
11-5-52	Posterior surface of epiglottis	Removed	Penicillin S-R	1 cc.	Daily	3 days	
1-5-53	Right false cord	Removed	Aureomycin Aureomycin	250 mgs. 250 mgs.	. . . . 4 × day	1 day, then 2 days	
1-21-53	Left true cord	Removed	Aureomycin	250 mgs.	4 × day	2 days	
2-25-53	Posterior right true cord	Removed	Aureomycin	250 mgs.	4 × day	3 days	and Podophyllum 10%, one application
3-26-53	Mid portion of right false cord; clearer than at any previous time	Removed	Aureomycin*	125 mgs.	4 × day	2 days	
5-20-53	Bilateral, but very small	Removed		125 mgs.	4 × day	2 days	
6-1-53	Left false cord	Removed	Aureomycin	50 mgs.	4 × day	2 days	
6-19-53	Improved; few small growths left false cord and epiglottis	Removed.					
7-15-53	Middle ⅔ of both cords	Removed					
8-11-53	Both cords	Removed, right					
9-2-53	None seen	. . . .					
10-2-53	Right true cord	Removed					

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* Cardiac arrest; chest opened and heart massaged.

Continued on Next Page

JLP, CONTINUED.

Date Examined or Treated	Condition Found. Location of Growths	Surgical Procedures	Antibiotics Given	Dosage	Frequency	Duration	Other Treatment
10-10-53	. . . .	. . . .	Crystalline Penicillin	1,200,000 U.	First day		
				400,000 U.	Daily	2 days	
				50,000 U.	4 × day	4 days	
11-20-53	Both false cords	Removed					
1-9-54	Both false cords	Removed					
2-12-54	Upper surface both cords and floor of ventricle	Removed					Podophyllum, 10%, one application
3-26-54	Multiple, bilat.	Removed	Penicillin	600,000 U.	Daily	2 days	
8-26-54	No papilloma seen	. . . .					
6-3-54	True and false cords, left side	Removed					
7-7-54	Ant. ⅓ left true cord, only	Removed					
10-22-54	Both cords	Removed					
11-10-54	Both cords	Removed					
1-26-55	Both cords	Removed					
2-23-55	Small, post. right true cord, only	Removed					
5-20-55	Left side clear; small on right	Removed	Penicillin S-R	600,000 U.	Daily	2 days	

Time covered by this record: Approximately five years. Age when last seen: 7 years. Condition when last seen: Greatly improved, but growths still recurrent.

DS FEMALE AGE 18 YEARS HOARSE FOUR YEARS AND PAPILLOMA REMOVED ELSEWHERE NINE TIMES SINCE 1948

Date Examined or Treated	Condition Found. Location of Growths	Surgical Procedures	Antibiotics Given	Dosage	Frequency	Duration	Other Treatment
10 3 50	Ant of left true cord ant commissure and a few on right side	Removed	Aureomycin	250 mgs	4 × day	3 days	
12 1 50	True cords normal						
2 7 51	Ant left true cord	Removed					
2 24 51	Left ventricle and left true cord	Removed	Penicillin	400 000 U	Daily	4 days	
2 30 51			Aureomycin	250 mgs	4 × day	5 days	
1 20-53	Hoarse, growth on epiglottis has been free since last admission until Oct 1952	Removed					
5 19 53	Left cord and epiglottis	Removed	Aureomycin	250 mgs	4 × day	6 days	
12 16 54	Ant left cord	Removed					
5 2 55	No gross papilloma						

Time covered by this record approximately five years Age when last seen 23 years Condition when last seen no gross papilloma voice good

ML, MALE. AGE 2 YEARS. HOARSE AND DYSPNEIC.

Date Examined or Treated	Condition Found. Location of Growths	Surgical Procedures	Antibiotics Given	Dosage	Frequency	Duration	Other Treatment
1-5-51	Entire glottic chink filled with papilloma	Cords were stripped	Aureomycin	125 mgs.	4 × day	5 days	
2-5-51	Left cord free, but return on right	Removed	Aureomycin	125 mgs.	4 × day	3 days	Podophyllum, 10%, one application
4-2-51	Recurrence, but to lesser degree	Removed					
Patient did not return.							

Time covered by this record: Three months. Age when last seen: 2 years. Condition when last seen: improved.

BAW, FEMALE. AGE 22 YEARS. HOARSE THREE WEEKS.

Date Examined or Treated	Condition Found. Location of Growths	Surgical Procedures	Antibiotics Given	Dosage	Frequency	Duration	Other Treatment
7-23-51	Ant. ½ both cords	Removed	Aureomycin	150 mgs.	4 × day	3 days	
8-10-51	Upper surface left true cord	Removed	Aureomycin Penicillin S-R	150 mgs. 400,000 U.	4 × day Daily	3 days 3 days	
9-6-50	Right true cord	Removed	Penicillin S-R	200,000 U.	Daily	3 days	
9-20-51	Much improved; small papilloma on right true cord	Removed	. . . .	. . . .	. . . .	. . . .	One application of podophyllum, 10%
10-4-51	Right cord	. . . .	Penicillin	200,000 U.	Daily	2 days	Electric cautery
10-25-51	Multiple, both cords	Removed	Aureomycin	100 mgs.	4 X day	2 days	. . . .
11-21-51	Entire length of left true cord	. . . .	Penicillin S-R	100,000 U.	Daily	2 days	Electric cautery
12-3-51	On both false cords; remainder of larynx now clear	Removed	Penicillin S-R	200,000 U.	Daily	2 days	
2-12-54	No papilloma	Examined only					

Time covered by this record: Approximately two and one-half years. Age when last seen: 25 years. Condition when last seen: larynx clear for two months.

GWR, MALE. AGE 9 YEARS. HOARSENESS.

Date Examined or Treated	Condition Found. Location of Growths	Surgical Procedures	Antibiotics Given	Dosage	Frequency	Duration	Other Treatment
8-3-52	Both cords and epiglottis covered	Removed					
12-29-53	Growths returned	Removed					
7-31-54	Growths returned	Removed					
12-20-54	Growths returned	Removed					
1-9-55	Growths returned	Removed					
1-19-55	Growths returned	Removed	Terramycin	125 mgs.	4 × day	3 days	
4-20-55	Ant. ⅓ left true cord, only	Removed					

Time covered by this record: Approximately 2 years. Age when last seen: 11 years. Condition when last seen: Greatly improved.

MH, FEMALE. AGE 41 YEARS. HOARSE FOR 10 YEARS.

Date Examined or Treated	Condition Found. Location of Growths	Surgical Procedures	Antibiotics Given	Dosage	Frequency	Duration	Other Treatment
5-1-53	Both true cords covered with papilloma	Removed					
6-12-53	Growths returned	Removed	Penicillin	400,000 U.	4 × day	3 days	
7-15-53	No recurrence	. . . .					
Patient did not return.							

Time covered by this report: 10 weeks. Age when last seen: 41 years. Condition when last seen: improved.

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PULSION DIVERTICULUM OF THE HYPOPHARYNX.*†

E. S. BRINTNALL, M.D. (By invitation),
E. L. GRANDON, M.D. (By invitation),
D. M. LIERLE, M.D., and
R. T. TIDRICK, M.D. (By invitation),

Iowa City, Ia.

Experience *in the management of pulsion diverticulum of* the hypopharynx in a series of 25 patients (1950-1955) has indicated that one-stage diverticulectomy is applicable in most patients. The method is safe in appropriate cases if a proper anatomical approach is utilized, and if the pharyngeal closure is secure and is accomplished without tension. Suspension of the sac and the two-stage excision is indicated in ill and malnourished patients, in the presence of inflammation and edema of the sac or following instrumental perforation of the diverticulum.

CLINICAL OBSERVATIONS.

Pulsion diverticulum of the hypopharynx is a protrusion of mucosal and submucosal layers of the pharynx through Killian's dehiscence[1] (between transverse fibers of the cricopharyngeus and oblique fibers of the inferior pharyngeal constrictor) into the posterior mediastinum. The most attractive theory of origin postulates incoordination of deglutition, with failure of the cricopharyngeus to relax in a manner appropriate to the second phase of swallowing. The weakest area of the pharynx (Killian's dehiscence) would be especially vulnerable to the abnormally increased intrapharyngeal pressure thus produced. Protrusion and diverticulum formation would be expected in this area of weakness, especially in the elderly.

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* Read at the Sixtieth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Montreal, Canada, May 16, 1956.

† From the Departments of Surgery and Otolaryngology, State University of Iowa, College of Medicine, and the Surgical Service, Veterans Administration Hospital, Iowa City, Ia.

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Twenty-one of this group of 25 patients were over the age of 60, and of these 18 were edentulous. The infrequency of occurrence of pulsion diverticula even in the edentulous, however, would suggest that poor mastication of food is not an important causative factor. Difficulty in swallowing is a common early symptom of these patients, but it is difficult to establish this symptom clearly as preceding the development of the sac. Three patients of this group on Roentgenologic study of a barium swallow had delay of entry of barium into the stomach. This might be evidence of incoordination of esophageal function. Though pulsion diverticula occur at a site of congenital weakness in the pharyngeal wall, they are considered to be acquired diverticula because their walls consist of only two of the three layers of the parent viscus (pharynx). We have seen only two examples of congenital diverticula and these were in newborn infants.[2]

The symptoms of pulsion diverticulum are related to the size of the hernial sac. When the sac is small the usual complaint is that of food sticking in the throat. When the sac is of moderate size it becomes dependent in position, fills easily with ingested material and leads to regurgitation. The very large diverticula fill with food and distort the hypopharynx and compress the esophagus, making it impossible for the patient to swallow an adequate amount of food. Malnutrition becomes increasingly more serious.

Lahey[3] proposed classification of these sacs into Stage I, Stage II and Stage III according to size and symptoms (see Fig. 1). The small (Stage I) sacs may annoy the patient, but they are not dangerous. The Stage II diverticula cause distressing regurgitation and gurgling and may be responsible for bronchitis or pneumonitis from inadvertent aspiration of contents of the sac. In Stage III sacs, malnutrition is added to the other difficulties.

Another hazard associated with the diverticula is possible instrumental perforation of the sac during diagnostic esophagoscopy or intubation. Great caution is necessary to avoid injury during this procedure. The esophagoscope passes easily into a large sac and the narrow slit-like aperture of the esophagus anterior to the large diverticular mouth is difficult to visualize. At times it is impossible to advance the instrument into the esophageal lumen. Mediastinitis may result from mucosal laceration without gross perforation of the diverticulum. A preliminary esophagram will establish the diagnosis of pulsion diverticulum and forewarn the endoscopist. Two patients in this series entered the hospital with mediastinitis following esophagoscopy. The first (T.B. No. 50-12190) was admitted five days following endoscopic perforation of the sac. He was treated by drainage of the posterior mediastinum and suspension of the sac, and he recovered. The second (P.H. No. 52-4198) was moribund from mediastinitis at the time of his readmission to the hospital three days following diagnostic esophagoscopy during which there was no gross perforation of the sac. He died the same day.

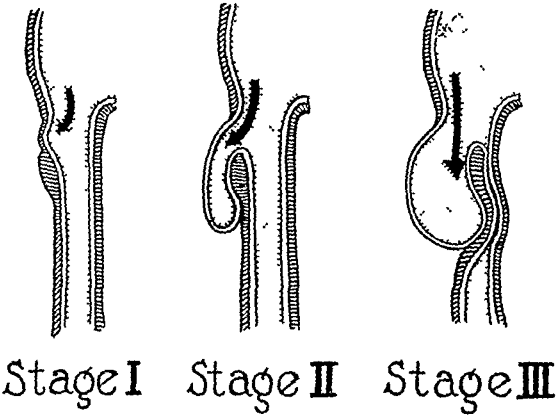

Fig 1 Diagram illustrating the three stages Stage I is symptomatic but does not interfere with swallowing or nutrition Stage II may cause annoying symptoms and be associated with regurgitation Stage III as illustrated is likely to be associated with mechanical interference with swallowing and resultant malnutrition

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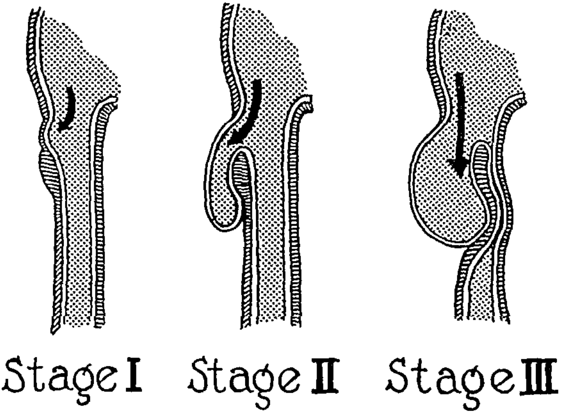

Fig. 1. Diagram illustrating the three stages. Stage I is symptomatic but does not interfere with swallowing or nutrition. Stage II may cause annoying symptoms and be associated with regurgitation. Stage III as illustrated is likely to be associated with mechanical interference with swallowing and resultant malnutrition.

Esophagoscopy is indicated preoperatively to remove solid material from the sac and to determine whether the sac is inflamed or ulcerated. A liquid diet is given before operation. In case of dehydration, appropriate fluid therapy is instituted. Transfusions of whole blood may be required if a lowered blood volume has resulted from malnutrition. Antibiotics are administered before operation if the diverticulum is inflamed or if there is pneumonitis.

Table I indicates the treatment carried out in the 25 patients seen during the years 1950-1955, inclusive. The stage of the sac is indicated in the same table. Sac extirpation was refused or was not advised in five patients. Three of these had Stage I protrusions. It is seen that the one-stage operation was most commonly employed, and usually for a Stage II sac. Suspension of the sac or two-stage excision was indicated in three patients because of (a) perforation of the sac with mediastinitis, (b) severe malnutrition, and (c) inflammation of the sac. Though the one-stage procedure is ordinarily preferred, staged operations are indicated if there is a threat of infection, if the patient's nutritional state is so poor that tissue healing is likely to be impaired, or if there exists a contraindication to anything more than simple elevation of the tip of the diverticulum.

TABLE I.

PULSION DIVERTICULA OF HYPOPHARYNX 1950-1955.

Treatment	Size (Stage) of Sac			Total
	I	II	III	
One stage excision	1	12	4	17
Two stage excision	0	0	1	1
Suspension of sac	0	1	1	2
Examination only	3	1	1	5
	4	14	7	25

There were two postoperative deaths in this group of patients. One death (H.H. No. 17975) occurred shortly following one-stage diverticulectomy from cardiac arrest. The second death (O.T. No. 51-14208) was due to carcinoma of the colon, and followed recovery from the first stage (sac suspension) procedure. The late results of operation were satisfactory. Recurrence of the diverticulum is known to have occurred in two patients (F.V. No. 50-10342 and D.S. No. 42-2764). In each patient the recurrent sac was small and non-symptomatic.

TECHNICAL CONSIDERATIONS.

Diverticulectomy is usually carried out under endotracheal anesthesia. In seriously ill patients, suspension of the tip of the sac by suture to the sternohyoid or sternocleidomastoid muscle can be accomplished with local and regional anesthesia.

Esophagoscopy is carried out in the operating room to make certain that the diverticulum is empty. At the same time, a Levine tube is threaded down the esophagus under direct vision if this can readily be done. If the Levine tube cannot be inserted before starting the operation, the anesthetist can easily introduce it after the diverticulum is mobilized and lifted cephalad. The excision and repair of the sac is carried out with the Levine tube in place. The tube may be removed at the termination of operation. It is left in place if there is indication for gastric or esophageal decompression, or need for early postoperative tube feeding.

Access to the posterior mediastinum is obtained through a transverse incision in skin at cricoid level. As this incision parallels lines of skin tension, it heals with minimal scarring, and the best possible cosmetic result is obtained. By placing the incision at this level, the level at which the neck of the diverticulum is located, and by elevating skin flaps in the plane between platysma myoides and deep cervical fascia, no restrictions are imposed upon deeper exposure. Deep fascia (Fascia colli) is incised along the anterior border of the sternocleidomastoid, extending as low as the clavicle if such exposure should appear to be required for the mobilization of a very large diverticulum. Approach is usually on the left side of the neck. After opening cervical fascia, the anterior infrahyoid muscles are retracted medially and the sternocleidomastoid muscle and the superior belly of the omohyoid are retracted laterally. As the lateral lobe of the thyroid gland is retracted forward and the carotid sheath lateralward, alar fascia is tensed between visceral and vascular compartments of the neck. The middle thyroid vein is ligated and divided as it crosses alar fascia. A vertical incision in alar fascia between superior and inferior thyroid arteries exposes the posterior mediastinum. In case of difficult mobilization of a large diverticulum, the inferior thyroid artery may have to be divided to allow dissection deeper into the posterior mediastinum.

The diverticulum is mobilized so that it can be lifted up. Care is exercised to avoid injury to the recurrent laryngeal nerves which ascend to the larynx close to the neck of the sac. The sac consists of mucosa and attenuated aponeurosis of pharynx but may have some muscle fibers near its neck. Such muscle bundles are swept off the neck of the sac. Care is taken to avoid over-zealous dissection at this point, as it is possible to mobilize pharyngeal mucosa to the extent of creating serious constriction of the pharynx and at the same time to produce dangerous tension at the line of suture used to close the pharynx after sac excision. As a further precaution against undue tension a stump of diverticular neck about one-fourth inch long is preserved, as this much tissue will be required for two lines of suture.

Ordinarily, a clamp is placed transversely across the neck of the sac, the sac is excised, and a continuous fine catgut suture is run over the clamp at the innermost suture line. This suture includes mucosal and submucosal layers of the pharynx. This inner line is then buried with a row of interrupted Lambert sutures of fine silk, which are placed in the aponeurosis of pharynx. This closure is then reinforced with interrupted silk sutures in muscularis of pharynx.

A Penrose drain is placed to the posterior mediastinum and closure of deep cervical fascia, platysma and skin follows.

SUMMARY.

1. Twenty-five patients with pulsion diverticulum of the hypopharynx were seen during the years 1950-1955.

2. One stage diverticulectomy is preferred for most patients. A transverse skin incision is advantageous. By leav-

ing a one-fourth inch cuff of diverticulum neck, secure closure is accomplished without tension and pharyngeal narrowing is avoided.

3. Sac suspension in the seriously debilitated or ill patient is the simplest and least hazardous procedure as a temporizing measure. *Staged excision of the sac is indicated if there is* local inflammation or severe malnutrition.

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3. Lahey, Frank H.: Esophageal Diverticula. *Surg. Clin. of N. A.*, 813-826, *June, 1953*.

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President: Dr. J. M. Tato, Azcuenaga 235, Buenos Aires, Argentina.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
General Secretary: Dr. C. E. Muñoz MacCormick, P. O. Box 9111, Santurce 29, Puerto Rico.
Meeting: Fifth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place:
President: Dr. J. H. Font, Medical Arts Bldg., San Juan, P. R.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis, Mo.
General Secretary: Dr. Paul Holinger, 700 No. Michigan Ave., Chicago (11), Ill.
Meeting: Statler Hotel, Washington, D. C., May 5-10, 1957.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

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Historian: Dr. Herman B. Cohen.
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Vice-President: Dr. Frank Wojniak, 6132 So. Kedzie Ave., Chicago, Ill
Secretary-Treasurer: Dr. Stanton A. Friedberg, 122 So. Michigan Ave., Chicago, Ill.
Meeting: First Monday of each Month, October through May.

OTOSCLEROSIS STUDY GROUP.

President: Dr. Gordon D. Hoople, 1100 East Genesee St., Syracuse, N. Y.
Secretary: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Meeting: Palmer House, Chicago, Ill., October, 1956.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
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President: Dr. Arthur S. Bristow, Princeton, Mo.
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THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. J. L. Levine.
Vice-Chairman: Dr. Russell Page.
Secretary: Dr. James J. McFarland.
Treasurer: Dr. Edward M. O'Brien.
Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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Chairman-Elect: Dr. Sherman B. Forbes, 706 Franklin Street, Tampa 2, Florida.
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Secretary: Dr. G. Slaughter Fitz-Hugh, 104 East Market Street, Charlottesville, Virginia.

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Howard L. Mitchell, Lexington, Va.
President-Elect: Dr. L. Benjamin Sheppard, 301 Medical Arts Building, Richmond 19, Va.
Vice-President: Dr. Edgar Childrey, Jr., Professional Building, Richmond, Va.
Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Va.
Annual Meeting:

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. W. L. Hughes, Lamar Life Bldg., Jackson, Miss.
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Meeting: George Vanderbilt Hotel, Asheville, N. C., Sept. 16-19, 1956.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Norman Eaddy, Sumter, S. Car.
Vice-President: Dr. J. H. Gressette, Orangeburg, S. Car.
Secretary-Treasurer: Dr. Roderick Macdonald, 333 East Main St., Rock Hill, S. Car.
Meeting jointly with the North Carolina Eye, Ear, Nose and Throat Society, George Vanderbilt Hotel, Asheville, N. Car., Sept. 17-18-19, 1956.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

esident: Dr. Chas. C. Grace, 145 King St., St. Augustine, Fla.
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THE GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

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Meeting: Quarterly, at Seven Seas Restaurant, February, May, October, and December.

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Chairman of Ophthalmology Section: Stephen J. Popovich, M.D.
Secretary of Ophthalmology Section: Richard Kratz, M.D.
Chairman of Otolaryngology Section: Harold Boyd, M.D.
Secretary of Otolaryngology Section: Howard G. Gottschalk, M.D.
Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles, California.
Time: 6:00 P.M., first Monday of each month from September to June inclusive—Otolaryngology Section. 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: H. Leroy Goss, M.D., 620 Cobb Bldg., Seattle 1, Washington.
Secretary-Treasurer: Homer E. Smith, M D, 508 East South Temple, Salt Lake City, Utah.
Meeting: April 7-11, 1957.

THE SOCIETY OF MILITARY OTOLARYNGOLOGISTS.

President: Col. Wendell A. Weller.
Secretary-Treasurer: Major Stanley H. Bear, M.C., 3810th USAF Hospital, Maxwell AFB, Alabama.
Time and place of meeting: October 16, 1956, Palmer House, Chicago, Ill.

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SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President: Dr. G. S. Gorrell, 110 Medical Arts Bldg., Calgary, Alta.
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Meeting:

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Meeting:

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Secretary:
Meeting: Lima, Peru, 1957.

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Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filho Dr. Penido Burnier and Dr. Gabriel Porto.

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Apartado Postal 21815
Mexico, D. F.

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(Nederlandsche Keel-Neus-Oorheelkundige Vereeniging.)

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Members: Dr. J. de Graaf, Dr. S. Muller, Dr. H. Navis.

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THE

LARYNGOSCOPE.

VOL. LXVI AUGUST, 1956 No. 8

A NEUROTOLOGIC DIFFERENTIATION BETWEEN HIGH AND LOW LYING DISTURBANCES OF THE CENTRAL NERVOUS SYSTEM.*

LYLE M. SELLERS, M.D.,

Dallas, Tex.

INTRODUCTION.

Prior to his death in 1934 Dr. E. R. Carpenter was the pioneer neurosurgeon of the Southwest. He had entered the field of neurosurgery after many years of active practice in ophthalmology and otolaryngology; consequently, he maintained an active interest in neurotology. Knowing of my primary interest in otology and the then young field of neurotology[1] I was privileged to see the majority of his cases that had neurotologic implications.

On March 24, 1931, we saw Mr R. V F., age 46, who complained of attacks of nonrotary vertigo with fulness of the ears but with no falling, staggering, emesis or sweating, beginning six months earlier. Placed on a restricted diet by his family physician he had improved. One week previously his attacks recurred, this time with "black outs", and on two occasions total loss of consciousness with subsequent periods of mental confusion. During one attack his physician had observed rigidity of the left arm with extension and fanning of the left fingers. He was now having hallucinations of smell.

On examination his hearing was within normal limits, and his equilibratory apparatus revealed no abnormal spontaneous phenomena. On rotation, with his head 30° forward and then 120° forward, all responses were normal. The Barany caloric tests with water at 20° C produced no response from either labyrinth. This was confirmed by the Kobrak test, using 10 cc of water at 5° C in each ear. Pneumoencephalograms and subsequent surgery revealed a large astrocytoma of the left temporal lobe.

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* Submitted as Candidate's Thesis to the American Laryngological Rhinological and Otological Society Inc 1956.

Editor's Note: This ms received in The Laryngoscope Office and accepted for publication January 20, 1956.

On November 16, 1933, we saw Mr. S. B., age 24. This patient complained of vertigo and staggering, of only one week's duration. He was growing worse rapidly. Hearing tests revealed slight right conduction deafness.

Examination of his equilibratory apparatus revealed spontaneous rotary nystagmus to the left of third degree. On left gaze it became severely oblique upward and left, and on upward gaze it became markedly vertical upward. On downward gaze it resumed its left rotary character. He past pointed six inches to the right with both hands. His gait was stiff and ataxic, there was a marked swaying Rombergism with his head in all the three positions; his pelvis was stiff, and his vertigo was disorganized.

On rotation to the right in both positions of the head his nystagmus was markedly exaggerated. It was of high amplitude and lasted 55 seconds. On rotation to the left his nystagmus was scarcely perceptible and lasted only 15 seconds. Both ears responded normally to the Barany caloric test with water at 20° C. A diagnosis of midline cerebellar tumor was confirmed at surgery.

Since several other cases seemed to support such a view I wondered whether these two cases had established a pattern of findings in high (probably supratentorial) and low (probably infratentorial) lesions.

By this time Dr. Carpenter's health was failing and he could no longer work with me on this problem. Other available neurosurgeons were uninterested in or antagonistic to the work, feeling that the otologist had little to offer, and that the rotation tests were actually dangerous to a patient with an intracranial lesion no matter how carefully they were done! Consequently my observations then became limited to the comparatively few and widely spaced cases that I encountered in my private otolaryngological practice. My interest did not flag; the problem was kept in mind, and whatever appeared in our journals concerning this condition was read and filed.

In 1951 I began making weekly trips to the United States Veterans' Administration Hospital at McKinney, Tex., in my capacity as consultant in otolaryngology to the Dallas and McKinney Hospitals. Since I was happy to see all cases indicating labyrinthine disturbance our department soon had as many such cases as we could handle.

Dr. Donald Corgill, the chief of service in otoloryngology at the hospital, became interested in these cases also. Since it is a field that receives but scant attention from so many clinicians, probably because of its time consuming nature, and so represented a new field of interest to them, our residents, most especially Drs. R. L. Duff and Jas. M. Anthony, became actively interested in this phase of otology. As these labyrinthine cases were worked out we discussed the anatomic and physiologic basis for our tests. At a general staff meeting, where our work in neurotology was being discussed, I mentioned my suspicion of the existence of high and low central nervous system lesion patterns and a possible anatomico-physiologic basis for such a pattern.

Dr. C. E. Kern, the chief of neurosurgery, also became interested. He is a neurosurgical student and a clinician of avid scientific interest, whose mind remains receptive to a new idea, even though suggested by an otologist. By his keen interest, his bitingly constructive criticism and his ready cooperation, we soon were able to see enough cases of suspected or already diagnosed intracranial disturbances (organic) to become of suggestive statistical significance.

It is on the basis of the original observation made 20 years ago, on the cases recently seen and reported here, and on reports in literature of scientific experimental work in neurology and neurotology by investigators of established ability and integrity, that this paper is written.

First, our method of examination will be described; then a report of a series of 52 consecutive cases seen in conjunction with the department of neurosurgery will be presented and analysed, and finally the anatomico-physiologic problem will be presented and developed to present a concept of the possible structures and pathways involved.

METHOD OF EXAMINATION.

A careful history is taken of all cases showing dysfunction of the equilibratory system. The patient is allowed to tell his own story, and then any pertinent questions are asked. This is followed by a physical examination of the ears, nose and throat.

A complete audiological examination, including high and low tone thresholds, the pure tone threshold curves by both air and bone conduction, speech reception ability and the presence or absence of recruitment and diplacusis is the first step of the actual examination of the cochleo-vestibular apparatus.

On November 16, 1933, we saw Mr. S. B., age 24. This patient complained of vertigo and staggering, of only one week's duration. He was growing worse rapidly. Hearing tests revealed slight right conduction deafness.

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This apparatus we hold to be a remote but integral part of the central nervous system, its upper limit being the cerebral cortex.

The examination of the canalo-vestibular portion of the cochleo-vestibular organ is begun by having the patient attempt to walk a straight line, with his eyes closed, to touch a point 20 feet ahead of him, observing his gait for steadiness, staggering or deviation.

We then observe his Romberg response with his face forward, to the right and to the left; his pelvic girdle reaction to sudden tilt to right, to left, backward and forward; the presence or absence of nystagmus with the head upright gazing straight ahead, to the right and to the left, then with the head tipped backward to a horizontal position while gazing upward to the ceiling, then with the head tipped laterally to the right shoulder and finally tipped laterally to the left shoulder while gazing on a level line. The presence or absence of past pointing is then established. His ability to detect rotary motion and its after-response is then observed by placing him in the turning chair and, with his eyes closed, turning him once slowly to the right and then after a slight pause, once slowly to the left.

The results of this portion of the examination we designate the spontaneous phenomena.

The classical Barany turning tests are then performed to determine at least nystagmus and post-rotary vertigo with the head tipped 30° forward. If any response calls for furthe turning, the head then is brought forward to the 120° position but here the rotation is at the speed of 10 turns in 20 second or one-half the speed with the head in the 30° position. Th change is made because the physical effect of centrifugal fo is so much enhanced by increasing the distance, equal to upper body length, from the center of rotation.

I consider the turning tests to be a *sine qua non* of th amination of the equilibratory apparatus. The normal tion of this apparatus is to respond to position and to m The turning tests probe this function; therefore they a the caloric tests are the true physiologic tests, and fai erform them must be accounted negligence.

The caloric tests, next in the order of the examination, formerly (prior to 1940) were carried out according to the Barany technique, using a continuous irrigation at 20° C. with the head 30° forward, and timing from the beginning of the irrigation to the onset of the nystagmus. Forty-five seconds was held to be normal. Decrease or increase of this time was held to indicate hyperactivity or hypoactivity; but the accuracy of these tests was subject to valid objections under certain conditions.

Because of its ease and accuracy I have changed to the Atkinson technique,[2] and in all cases since 1940 I have used this method as the basic caloric test in the following manner:

A small pan is filled with chipped ice and a small quantity of tap water is added. Within a matter of moments this system of melting ice attains a temperature of 0° C. This temperature will maintain itself if the pan is kept covered until the ice has almost completely melted. A 1 cc. tuberculin syringe (barrel and plunger separated) is placed in this solution and allowed to remain for about five minutes. Due to the poor heat conducting quality of glass and to the relatively thick wall of the syringe in relation to the small volume of water in the barrel the temperature of the water in the filled syringe will not change in the very few seconds required to convey it from the pan to the patient's ear.

The patient is tipped to the left until the head is in the left lateral horizontal position and then the chin is rotated upward about 30°, which brings the horizontal canal into position for a maximum response, as it is the horizontal and not the vertical canal which responds to this test producing the initial rotary vertigo. The patient then is instructed to gaze at a point straight ahead and is warned not to move when the cold water is instilled into the ear. The chilled syringe is filled with 1 cc. of the water at 0° C. and promptly discharged into the external auditory canal. A split second two-handed stop watch is started at the same instant, and the eyes of the patient are watched intently for the first evidence of nystagmus. As experience is required to make this observation with accuracy the resident is required to do this with me using his own stop watch. After a surprisingly few number of observations we rarely will differ by more than a fraction of a second in spotting the first twitch. As soon as this first twitch occurs the first of the two hands of the stop watch is stopped to signal the end of the latent period. As soon as we are assured that the nystagmus has started and its direction has been noted the patient is brought to the upright (head 30° forward) position allowing the water to drain from the ear, the eyes are closed, and the presence or absence of past pointing is established. This tests the reactions commonly ascribed to the vertical canals. The head is then tipped backward to a horizontal position, the eyes are opened, and the patient is instructed to look at the ceiling. The direction and the degree of the nystagmus are observed again. The eyes are closed and the past pointing reaction is observed in this position. This tests the reaction commonly ascribed to the horizontal canal. The head is returned to the upright position, the eyes are opened and fixed on a point straight ahead and the nystagmus is watched until it ceases. At this instant the second of the two hands of the stop watch is stopped. The time thus recorded, minus the latent period, represents the duration of the nystagmus.

In normal cases the ratio of the latent period to the duration is established quite constantly as 1 : 4. This ratio we speak of as the Atkinson ratio.[2] It is the ratio and not the time that is important. The average normal latent period we have found to be about 20 seconds, and the duration time to be about 80 seconds or 1 : 4; but a latent period of 15 seconds and a duration time of 60 seconds still would be normal, as would a latent period of 25 seconds and a duration time of 100 seconds.

If the ratio is altered it indicates altered action of the equilibratory apparatus. Shortening of the latent period in relation to the duration time, *e.g.*, 15 seconds to 75 seconds, or 1 : 5, denotes hyperactivity or irritation. Lengthening of the latent period in relation to the duration time, *e.g.*, 25 seconds to 75 seconds, or 1 : 3, indicates hypoactivity by suppression or blocking.

The patient is allowed to rest for about 15 minutes, or at least until all evidence of reaction to the test has subsided. The left ear then is tested in the same manner. All caloric tests in the cases herein reported were performed according to this technique. So accurate and so easily mastered is this test that at least three correct localizations in these cases were made by the resident staff in cases that required an immediate report at a time that I was unavailable.

The results obtained by the rotational and the caloric tests we designate as the induced phenomena.

REPORT AND ANALYSIS OF CASES.

With the exception of the two cases mentioned in the Introduction, which are not included here, and the last case to be reported which was seen by me fortunately, as well as fortuitously, in our private practice just as the series was closed, all the cases were at the U. S. V. A. Hospital at McKinney, Tex. All are white males. They are all the cases seen in continuous chronological series (correctly and incorrectly localized or undiagnosed) in conjunction with the department of neurosurgery; therefore they are unselected and are controlled as to diagnosis.

The cases are numbered in the chronological order in which we saw them, but for convenience in presentation they have been regrouped as to localization where localization was correct, and as to incorrect and unconfirmed localizations. The case numbers as presented in the report do not appear in the order in which they were seen, but they are all present—from Case No. 1, seen August 19, 1951, to case No. 52, seen privately, June 18, 1955.

Cases confirmed by neurosurgical, neurological or medical diagnosis with their localization are as follows:

Those numbered 6, 7, 9, 18, 28, 33, 34, 45, 49 and 52 are high, localized lesions.

Nos. 2, 35, 10, 1, 16, 25, 26, 31, 50 and 44 are high, diffuse lesions.

Nos. 5, 40 and 30 are low lesions, localized and diffuse.

Nos. 37, 46 and 38 are low, localized lesions with high extension.

Nos. 3, 4, 14, 19, 29, 32, 43, 47 and 27 are lesions located near the tentorium, with either high or low extension.

Nos. 11, 12, 17, 22, 24, 39 and 42 are diffuse lesions with widespread generalized high and low symptoms.

Definite errors in diagnosis or cases that were temporarily unconfirmed by neurosurgical or neurological opinion are numbered 8, 13, 15, 21, 23, 36 and 41. Those numbered 20, 28 and 51 are lesions of the cerebello-pontine angle.

Case No. 6: J. B. F., age 39. Admitted March 5, 1952. Examined March 14, 1952. Reg. No. 34108.

History: In October, 1949, this patient suffered a severe headache, rotary vertigo and pain and limp in the left leg. Diagnosed at Brook General Hospital as "cyst of the third ventricle." Nothing was done at this time. On May 21, 1951, he entered this hospital and the case diagnosed as supracellar cyst or craniopharyngeoma; inoperable. The patient left this hospital and a private neurosurgeon did a Thorkildsen procedure. This was followed by severe aseptic meningo-encephalitis. On March 5, 1952, he returned to this hospital with residual symptoms indicating "bilateral pyramidal and extra-pyramidal disease indicating affection of both basal ganglia and internal capsules, perhaps also involving the hypothalamus."

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

Head 30° Forward.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 22 secs.	Hor. to right. *Amp.*—Good; Dur. 22 secs.
Vert.—Good; no autonomic phenomena.	Good; no autonomic phenomena.

Head 30° Forward.

D. *Caloric*—Right ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good; After 55 secs. Dur. 50 secs.	Rot. to right. *Amp.*—Good; After 35 secs.; Dur. 62 secs.

Head Back.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Good.
Atkinson Ratio—1:1-;	1:2-,

Summary—Response to rotation is normal; response to caloric test markedly subnormal.

Neurosurgical Diagnosis—Supracellar cyst.
Meningo-encephalitis with residual pyramidal and extrapyramidal tract involvement.

Case No. 7: L. G., age 30. Examined March 20, 1952. Reg. No. 34566.

History: This patient was operated upon in this hospital on September 18, 1951. At that time a deep lying astrocytoma was found in the right

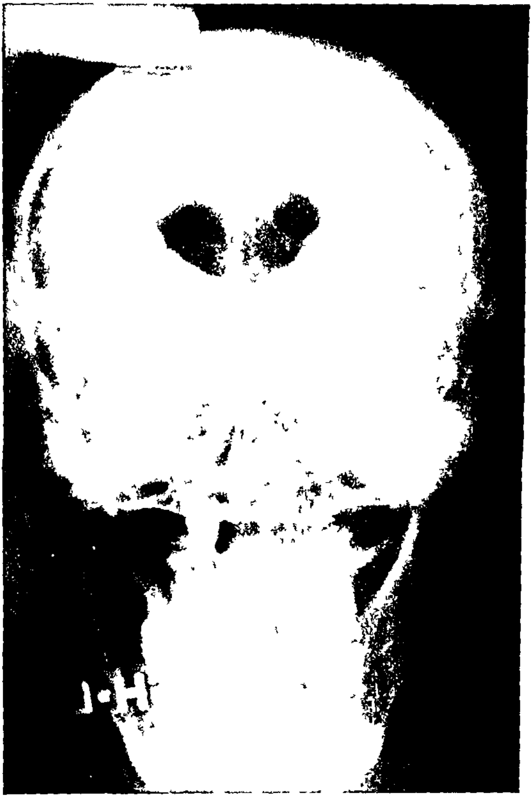

Fig. 1. Encephalogram of Case No. 6 showing marked dilation of the lateral ventricles from blocking in the third ventricle.

temporal lobe. He was sent to the department of otolaryngology without history for our evaluation.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

Head 30° Forward

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 27 secs.	Hor. to right. *Amp.*—Good; Dur. 27 secs.
Vert.—Good; no autonomic phenomena.	Good; sweat and pallor.

Head 120° Forward

Nystagmus—Rot. to left. *Amp.*—Good; Dur. 17 secs.	Rot. to right. *Amp.*—Good; Dur. 15 secs.
Falling—To right; good.	To left; good.
Vert.—Good; pallor and sweat.	Good; pallor and sweat.

Head 30° Forward

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—None.	Rot. to right. *Amp.*—Good after 26 secs.; Dur. 92 secs.
Vert.—Subnormal; pallor and sweat.	Subnormal pallor and sweat.

Head Back.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Good.
Vert.—Subnormal.	Subnormal.
Atkinson Ratio—Not Obtainable.	1:3.5.

Summary—Response to rotation normal; right vertical tracts completely blocked; left vertical tracts subnormal to caloric test.

Case No. 9: W. L. H., age 30. Admitted April 14, 1952. Examined April 18, 1952. Reg. No. 34947.

History: Two and one-half weeks ago upon straightening up after a backward bent position while at work, patient suffered a severe attack of pain and numbness in the left occiput, neck, shoulder, arm and left chest. The left hand failed to grasp objects properly; *i.e.* he did not place his hand in accordance with the shape of an object (thalamic symptom?).

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

Head 30° Forward.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 24 secs.	Hor. to right. *Amp.*—Good; Dur. 28 secs.
Vert.—Good; slight pallor and sweat.	Good; slight pallor and sweat.

Head 120° Forward

Nystagmus—Rot to left *Amp*—Good, Dur 15 secs	Rot to right *Amp*—Good, Dur 15 secs
Falling—Good to right	Good to left
Vert—Good, pallor, sweat and nausea	Good, pallor, sweat and nausea

Head 30° Forward

D *Caloric*—Right ear	Left ear
Nystagmus—Rot to left *Amp*—Fair, After 35 secs, Dur 90 secs	Rot to right *Amp*—Fair, After 37 secs, Dur 78 secs
Vert—Marked, pallor, sweat and emesis	Good, slight nausea

Head Back

Nystagmus—Hor to left *Amp*—Fair	Hor to right *Amp*—Fair
Vert—Good	Good
Atkinson Ratio—1 2 6	1 2

Summary—Response to rotation normal response to caloric test markedly subnormal especially on the left

Neurotologic Diagnosis—A high central lesion, probably in the region of the thalamus

Neurosurgical Diagnosis—A vascular accident in the upper brain stem (mesencephalic) region up to the lower thalamic nuclei

Case No 18 W E A, age 32 Admitted November 20, 1952 Examined November 21 1952 Reg No 39370

History Pain in the right ear and mastoid and in the left frontal area for one month Since then increasing diplopia, nausea and staggering

EXAMINATION

A *Hearing*—A moderate bilateral *conductive* type impairment

B *Spontaneous Phenomena*—Normal except a deviation of 5 feet to the left in walking the 20 feet line

Head 30° Forward

C *Rotation*—To right	To left
Nystagmus—Hor to left Amp—Good Dur 25 secs	Hor to right *Amp*—Good, Dur 27 secs
Vert—Fair, little or no autonomic phenomena	Fair, moderate autonomic phenomena

Head 30° Forward

D *Caloric*—Right Ear	Left ear
Nystagmus—Rot to left *Amp* Poor, After 15 secs, Dur 85 secs	Rot to right *Amp*—Small After 30 secs, Dur 88 secs
Vert—Good, nausea, pallor and sweat	Fair, subnormal autonomic phenomena

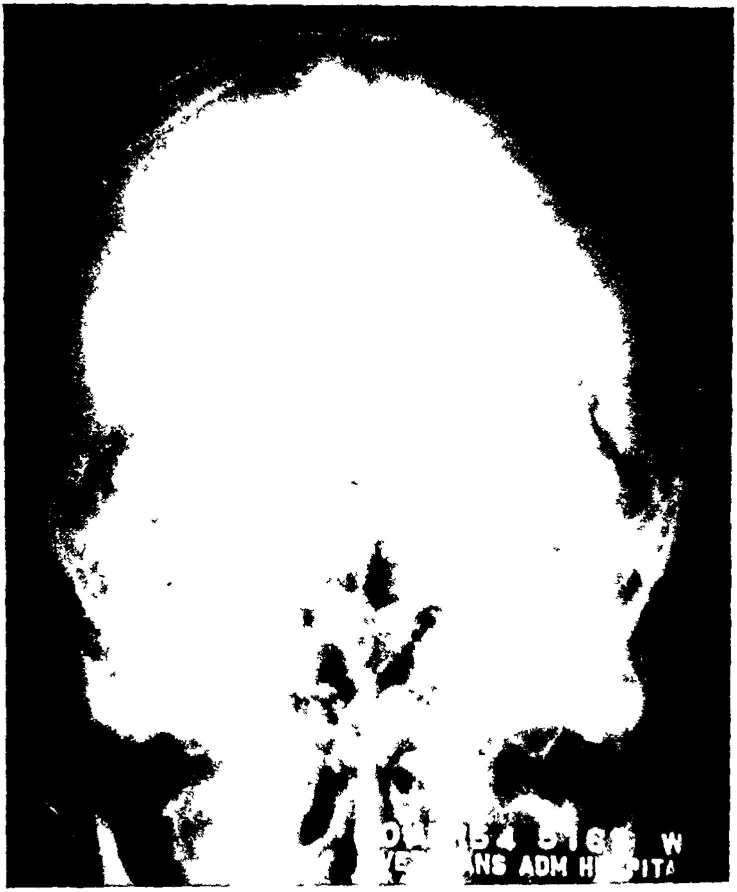

Fig. 3 Case No. 33. Anterior-posterior encephalogram showing dilated ventricles with tumor of the third ventricle

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 30 secs.	Hor. to right. *Amp.*—Good; Dur. 28 secs.
Vert.—Good; marked pallor and sweat.	Good; marked pallor and sweat.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Fair after 43 secs.; Dur. 102 secs.	Rot. to right. *Amp.*—Poor after 47 secs.; Dur. 104 secs.
Vert.—Little or none; no autonomic phenomena.	Little or none; no autonomic phenomena.

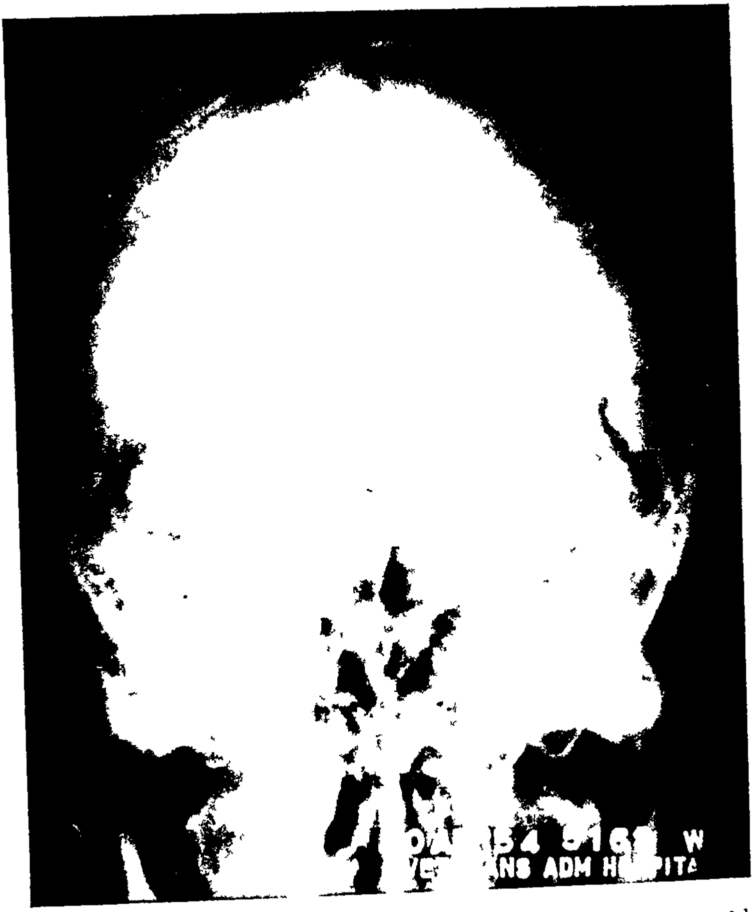

Fig. 3 Case No 33 Anterior-posterior encephalogram showing dilated ventricles with tumor of the third ventricle

Head 30° Forward.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 30 secs.	Hor. to right *Amp*—Good; Dur. 28 secs.
Vert—Good, marked pallor and sweat.	Good, marked pallor and sweat.

Head 30° Forward.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Fair after 43 secs.; Dur. 102 secs.	Rot. to right. *Amp.*—Poor after 47 secs.; Dur. 104 secs.
Vert—Little or none; no autonomic phenomena	Little or none; no autonomic phenomena.

HEAD BACK

Nystagmus—Hor to left *Amp* Fair	Hor to right *Amp*—Poor
Vert—Little or none	Little or none
Atkinson Ratio—1 2 4	1 2 2

Neurotologic Diagnosis—Lesion high—probably floor third ventricle near hypothalamus

Encephalogram—Dilated lateral ventricles with tumor anterior portion third ventricle

Neurosurgical—On May 10, 1954, a massive tumor (malignant adamantinoma) filling the third ventricle

Comment—This tumor was beginning to exert pressure on the posterior fossa structures

CASE No 34 R S J, age 31 Admitted September 27, 1954 Examined October 15, 1954 Reg No 52679

History This patient complained of nervousness indecisiveness head aches (much worse during the past five years), parosmia and increasing inability to think There had been one recent attack of loss of consciousness

EXAMINATION

A *Hearing*—Within normal limits

B *Spontaneous Phenomena*—A first degree nystagmus, greater on looking to the right than to the left Otherwise within normal limits

HEAD 30° FORWARD

C *Rotation*—To right	To left
Nystagmus—Hor to left *Amp* Good Dur 25 secs	Hor to right *Amp*—Good Dur 23 secs
Vert—Good slight pallor and sweat	Good, no autonomic phenomena

HEAD 30° FORWARD

D *Caloric*—Right Ear	Left Ear
Nystagmus—Rot to left *Amp*—Small after 31 secs, Dur 84 secs	Rot to right *Amp*—Small after 34 secs Dur 83 secs

HEAD BACK

Vert—Fair	Fair
Atkinson Ratio—1 2 7	1 2 5

Summary—Responses to rotation normal Responses to caloric tests markedly depressed

Neurotologic Diagnosis—A high central lesion

Encephalogram and Arteriogram—A tumor in the right frontal region —Left hydrocephalus

Neurosurgical—On October 18, 1954, a large astrocytoma was found at operation in the infero medial half of the right frontal lobe

CASE No 45 S D M, age 26 Admitted March 25, 1955 Examined April 15 1955 Reg No 56368

History On October 10, 1954, this man suffered with a transient roaring throughout his entire head Since that time he has had nine such attacks, each accompanied with dimness of vision and followed by numbness of the left arm and leg. Otherwise his history is completely negative.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 28 secs.	Hor. to right. *Amp.*—Good; Dur. 27 secs.
Vert.—Good; no autonomic phenomena.	Good; no autonomic phenomena.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Fair after 52 secs.; Dur. 83 secs.	Rot. to left. *Amp.*—Fair after 16 secs.; Dur. 103 secs.
Vert.—Good; sweat.	Good; sweat, pallor and nausea.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to left. *Amp.*—Good.
Vert.—Good.	Good; sweat, pallor and nausea.
Atkinson Ratio—1:1.6.	1:6.4.

Summary—Response to rotation normal. Responses to caloric test show right markedly depressed; left markedly irritated.

Neurotologic Diagnosis—High blocking (primary) lesion on right; high irritation left.

Encephalograms—Ventricular dilatation and tumor in the right frontal lobe.

Neurosurgical Diagnosis—Operation on April 18, 1955, revealed a glioma in the posteroinferior portion of the right frontal lobe.

CASE No. 49: H. T. C., age 49. Admitted May 25, 1955. Examined June 1, 1955. Reg. No. 57716.

History: This normally loquacious right-handed individual was entirely normal until three weeks ago, when he became morose and disinclined to talk. One week ago he became unable to speak intelligibly, uttering only mumbling sounds. There was some vomiting of a profuse mucoid material.

EXAMINATION.

There was hemiparesis generalized on the right including all branches of the facial nerve; also a marked nominal aphasia although he understood perfectly, carrying out all directions as we performed the tests on his ears.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—Except deviation to the right of six feet (a result of this hemiparesis?) all were within normal limits.

HEAD 30° FORWARD

C *Rotation*—To right	To left
Nystagmus—Hor to left *Amp*—Good, Dur 29 secs	Hor to right *Amp*—Good, Dur 27 secs
Vert—Good, nausea and retching	Good, sweat and emesis

HEAD 30° FORWARD

D *Caloric*—Right Ear	Left Ear
Nystagmus—Rot to left *Amp*—Good after 20 secs, Dur 107 secs	None
Vert—Marked, emesis, pallor and sweat	Marked, emesis, pallor and sweat

HEAD BACK

Nystagmus—Hor to left *Amp*—Good	Hor to right *Amp*—Good
Vert—Marked	Good
Atkinson Ratio—1 53	Not obtainable

Neurotologic Diagnosis—High lesion on left involving Broca s area

Summary—All responses normal except from vertical tracts on left to caloric test

X-ray of Chest—Inoperable carcinoma (bronchogenic) left upper pulmonary lobe

Final Diagnosis—Metastasis bronchogenic carcinoma to Broca's area left frontal lobe

Patient expired in this hospital July 12, 1955 Autopsy revealed metastatic tumor in left temporal lobe

CASE No 52 G B D, age 41 Private practice Seen and examined June 18 1955

History This man was operated on in 1925 by Dr Carpenter, who removed a suprasellar cyst The approach was right temporal area Since surgery he has been totally blind and partially paralyzed in the left arm and leg His hearing so far as he knew was normal About two years ago he awakened one morning to find himself totally deaf in the left ear He does not recall any unusual dizziness at that time

EXAMINATION

A *Hearing*—Right ear normal, left ear total deafness

B *Spontaneous Phenomena*—Nystagmus first degree on right and left lateral gaze Vertical on upward gaze Otherwise normal

HEAD 30° FORWARD

C *Rotation*—To right	To left
Nystagmus—Hor to left *Amp*—Poor, Dur 15 secs	Hor to right *Amp*—Poor, Dur 15 secs
Vert—Poor	Poor

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Obl. to left. *Amp.*—Huge after 15 secs.; Dur. 130 secs.	None.
Vert.—Good; sweat.	Questionable; no autonomic phenomena.

HEAD BACK.

Nystagmus—Obl. to left. *Amp.*—Huge irregular.	None.
Vert.—Good.	None.
Atkinson Ratio—1:8.6.	

Summary—On rotation the canals respond as with one labyrinth dead and compensated; on caloric test the right ear shows marked hyperactivity—central irritation.

Diagnosis—Postoperative high central irritation; total occlusion left cochleo-vestibular artery.

CASE NO. 2: J. P. H., age 59. Admitted November 3, 1951. Examined November 30, 1951. Reg. No. 31728.

History: For the past six years this patient had suffered a persistent and progressive sense of imbalance and vertigo with staggering.

EXAMINATION.

A. *Hearing*—Within normal limits for age.

B. *Spontaneous Phenomena*—A tendency to backward sway in all three positions of the head, and a spastic gait; otherwise normal.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 25 secs.	Hor. to right. *Amp.*—Good; Dur. 23 secs.
Vert.—Good; no autonomic phenomena.	Good; slight nausea.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—None.	Rot. to right. *Amp.*—Small after 27 secs.; Dur. 92 secs.
Vert.—None.	Poor.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Very small.
Vert.—Little or none.	Little or none.
Atkinson Ratio—..........	1:3.4.

Summary—Responses to rotation are normal; responses to caloric testing show none from the right vertical tracts, subnormal from the left vertical tracts, normal from the right horizontal tracts and markedly subnormal from the left horizontal tracts.

Neurotologic Diagnosis—A diffuse process, probably vascular, high in the brain stem.

Neurologic Diagnosis—Diffuse cerebral arteriosclerosis.

CASE No 35 H H, age 40 Admitted October 8, 1954 Examined October 22, 1954 Reg No 52941

History This patient complained of "numbness" of the left side of his face Three weeks previously he suddenly fell to the ground, his neck became stiff, and he quickly lapsed into unconsciousness He remained unconscious for 24 hours and was left with the numbness and weakness of the left side of the face, and with a limp of the left leg Spinal tap at the time of the attack revealed blood in the fluid

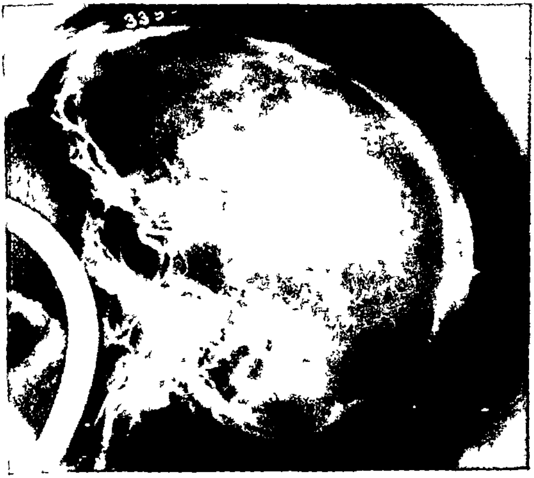

Fig 4 Case No 35 Right carotid arteriogram with no conclusive demonstration of a normal anterior cerebral artery

EXAMINATION

There was a subtotal paralysis of the left facial nerve typical of a supranuclear lesion

A *Hearing*—Within normal limits

B *Spontaneous Phenomena*—All within normal limits

HEAD 30° FORWARD

C *Rotation*—To right	To left
Nystagmus—Hor to left *Amp*—Large, Dur 28 secs	Hor to right *Amp*—Large, Dur. 25 secs
Vert—Good, profuse sweat, severe emotional reaction—crying	Good, profuse sweat, less emotional reaction

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good after 37 secs.; Dur. 99 secs.	Rot. to right. *Amp.*—Fair after 43 secs.; Dur. 72 secs.
Vert.—Fair.	Fair.

HEAD BACK.

Nystagmus—Rot. to left. *Amp.*—Good.	Rot. to right. *Amp.*—Fair.
Vert.—Fair.	Fair.
Atkinson Ratio—1:3.	1:1.6.

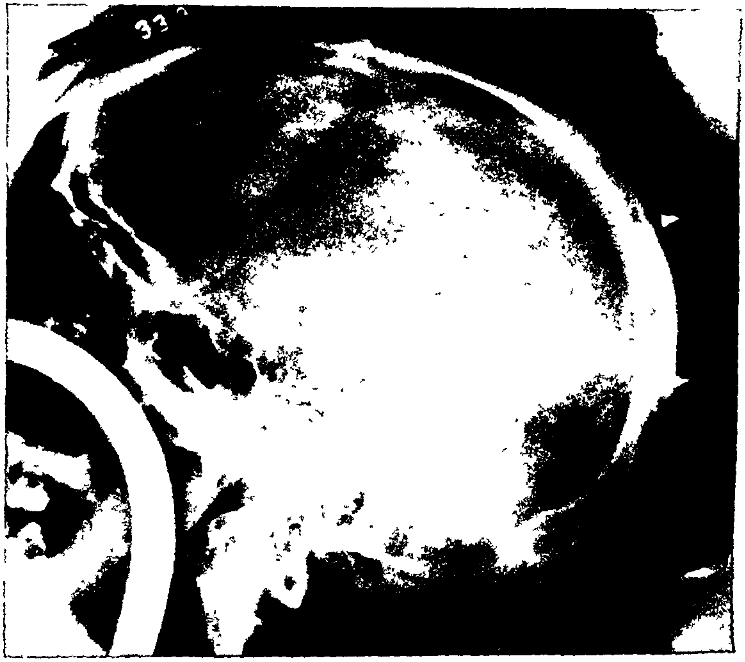

Fig. 5. Case No. 35. Right vertebral arteriogram showing only the vertebral and the beginning of a basal artery

Summary—Responses from rotation are normal; responses from caloric tests are subnormal on the right and markedly subnormal on the left.

Neurotologic Diagnosis—A high stem lesion on the left.

Arteriograms—These show *1.* A relatively small right carotid artery with no conclusive demonstration of a normal anterior cerebral artery on the right side (the vascular system is atrophic or anomalous). *2.* The left vertebral artery angiogram shows only the vertebral and the beginning of a basilar artery.

Neurosurgical Diagnosis—A subarachnoid hemorrhage in the region of the basilar artery

Case No 10 W O S, age 52 Admitted May 5, 1952 Examined May 9, 1952 Reg No 35373

History This patient has had a suboccipital headache, persistent for ten or twelve years Since 1950 he has had episodic severe pain in the left eye He says the hearing in the left ear is impaired

EXAMINATION

The left pupil is smaller than the right Weakness of the left palate and uvula with diminished gag reflex on the left Diminished corneal reflex on the left and diminished taste on the anterior two thirds of the tongue on the left Diminished sense of touch in all branches of the fifth nerve There is weakness of the left hand The blood pressure is 250/150

A *Hearing*—The right ear is normal for his age, the left ear shows moderate downward curve loss in the upper registers

B *Spontaneous Phenomena*—His gait was ataxic and he tended to fall to the left in the Romberg test in all positions of the head, otherwise within normal limits

HEAD 30° FORWARD

C *Rotation*—To right	To left
Nystagmus—Hor to left *Amp*—Good, Dur 27 secs	Hor to right *Amp*—Good, Dur 32 secs
Vert—Good, slight nausea and sweat	Good, no autonomic phenomena

HEAD 30° FORWARD

D *Caloric*—Right Ear	Left Ear
Nystagmus—Rot to left *Amp*—good, After 43 secs, Dur 96 secs	Rot to right *Amp*—Good, After 30 secs, Dur 99 secs
Vert—Fair, nausea	Good, nausea, emesis, profuse sweat

HEAD BACK

Nystagmus—Hor to left *Amp*—Good	Hor to right *Amp*—Good
Vert—Fair	Good
Atkinson Ratio—1 2 2	*1 3 3*

Summary—Responses via the tracts from the right ear to rotation are exaggerated, responses to the caloric test are depressed, markedly so from the right ear

Neurotologic Diagnosis A high diffuse lesion most marked on the right side

Neurosurgical Diagnosis An undiagnosed intracranial lesion as the patient refused to permit an arteriogram or encephalogram

In April, 1955, he returned to the neurosurgical service for re-evaluation At that time the diagnosis was degenerative central nervous system disease probably syringomyelia

On May 17, 1955, he committed suicide by shooting himself in the head because of unbearable headache

Case No. 1: A. G. G., age 40. Admitted August 13, 1951. Examined August 17, 1951. Reg. No. 30027.

History: This patient complained of pain on the right side of the body (typical Sluder's syndrome to ear, neck, shoulder, arm and leg), inability to have an erection, although he achieves orgasm without erection; depression, "don't give a damn"; contemplating suicide, and illusions of persecution involving, especially, his wife. Since childhood he states that he passes out easily but without convulsions. In 1931 he was injured in an auto wreck, being unconscious for two hours. In 1941 he was struck on the chin with an eight-pound sledge hammer and was unconscious for one hour.

EXAMINATION.

Negative except decreased sensation to pin prick on right forehead and scalp and on the left shoulder and arm.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

Head 30° Forward.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 24 secs.	Hor. to right. *Amp.*—Good; Dur. 22 secs.
Vert.—Good; no autonomic phenomena.	Good; pallor, sweat and nausea.

Head 30° Forward.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—None.	None.
Vert.—Fair; pallor, sweat and nausea.	Fair (delayed); subnormal autonomic phenomena.
Atkinson Ratio—...........	....

Head Back.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Good.
Vert.—Good.	Fair.

Summary—Responses to rotation normal; responses from vertical tracts bilateral absent from caloric stimulation.

Neurotologic Diagnosis—A central cortical or high stem lesion.

Neurosurgical Diagnosis—A predisposition, severe, with a marked obsessive-compulsive character developed in a reaction to hostile mother fantasies and defects of emotional control and conceited intellectual performances. All probably residual to a childhood influenzal encephalitis or to severe head injuries during adolescence.

Case No. 16: H. M. S., age 22. Admitted October 21, 1952. Examined October 31, 1952. Reg. No. 38770.

History: Twelve hours previously there was a sudden severe headache, bifrontal and radiating to both temples with hallucination of vision in the left eye. This was followed quickly by bilateral blindness, mania and unconsciousness.

EXAMINATION

A *Hearing*—The audiogram showed a bilateral drop to 35 db at 4,000 cps Otherwise within normal limits

B *Spontaneous Phenomena*—A large horizontal nystagmus to the left, especially on slight *right* lateral gaze On far right lateral gaze this changed to first degree to the right In the back horizontal position of the head there was a rotary nystagmus to the left Otherwise all within normal limits

HEAD 30° FORWARD

C *Rotation*—To right	To left
Nystagmus—Hor to left *Amp*—Good Dur 30 secs	Hor and Rot to right *Amp*—Good, Dur 43 secs
Vert—Subnormal, no autonomic phenomena	Subnormal, no autonomic phenomena

HEAD 30° FORWARD

D *Caloric*—Right Ear	Left Ear
Nystagmus—Rot to left *Amp*—Good, After 18 secs, Dur 102 secs	Rot to right *Amp*—Huge after 22 secs, Dur 158 secs
Vert—Little or none	Little or none

HEAD BACK

Nystagmus—Hor to left *Amp*—Good	Hor to right *Amp*—Good
Vert—Little or none	Little or none
Atkinson Ratio—1 5 6	1 7 2

Summary—Responses to rotation show moderate hyperactivity especially from the right tracts, caloric test shows marked hyperactivity especially from the left tracts

Neurotologic Diagnosis—Cerebral or high stem irritation with left cerebellar involvement

Neurosurgical Diagnosis—Acute brain syndrome (cerebral vascular occlusion)

CASE No 25 M D R, age 42 Admitted September 20, 1953 Examined October 30, 1953 Reg No 45365

History This patient complained of attacks of severe headache with loss of consciousness noted especially just as he would be falling asleep There is a moderate diplopia which caused a recent car wreck Condition progressive over several years

EXAMINATION

A *Hearing*—Within normal limits

B *Spontaneous Phenomena*—Deviated 3 feet to the left Otherwise all within normal limits

HEAD 30° FORWARD

C *Rotation*—To right	To left
Nystagmus—Hor to left *Amp*—Good Dur 21 secs	Hor to right *Amp*—Good, Dur 23 secs
Vert—Fair, no autonomic phenomena	Fair to good no autonomic phenomena

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good; After 22 secs.	Rot. to right. *Amp.*—Fair after 35 secs.
Vert.—Fair to good.	Poor to fair.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Fair.	Hor. to right. *Amp.*—Good.
Vert.—Poor.	Good.
Atkinson Ratio—1:5.2.	1:2.8.

Summary—Responses to rotations are normal; to the caloric test, hyperactivity on the right and marked hypoactivity on the left.

Neurotologic Diagnosis—A high central lesion, nature unknown.

Encephalogram—Cerebral atrophy.

Neurosurgical Diagnosis—Post-traumatic cerebral atrophy with traumatic epilepsy.

CASE NO. 26: L. R. G., age 35. Admitted October 24, 1953. Examined November 6, 1953. Reg. No. 46057.

History: In September this soldier suffered a shell injury to the left fronto-temporal area. In February, 1945, a metal plate was set at this site. Since that time he has had persistent dull aching bilateral frontal headache, partially relieved by aspirin. There were also occasionally mild episodes of dizziness with vomiting. Two weeks ago there was an episode of severe headache with dizziness and sub-total loss of consciousness.

EXAMINATION.

A scar in the left fronto-parietal area where X-ray shows a metal plate. The eye grounds show a yellowish area with a small amount of pigmentation. in the bilateral superior temporal areas. There was a moderate emotional flattening with memory deficit.

A. *Hearing*—Normal except that the audiogram showed a bilateral nicking at 5792 cps. on the right to 25 db and on the left to 60 db.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 24 secs.	Hor. to right. *Amp.*—Good; Dur. 23 secs.
Vert.—Good; sweat and nausea.	Good; pallor, sweat and nausea.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—None.	Rot. to right. *Amp.*—Large; after 42 secs.; Dur. 105 secs.
Vert.—Fair.	Good; nausea and emesis.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Large.	Hor. to right. *Amp.*—Huge.
Vert.—Marked. Sweat, nausea and emesis.	Marked. Sweat, nausea and emesis.
Atkinson Ratio—............	1:2.5.

Summary—Responses to rotation are normal, caloric test shows absence of response from right vertical tracts, hypoactivity from the left vertical tracts, and marked hyperactivity from both horizontal tracts

Neurotologic Diagnosis—Posttraumatic high central lesion

Neurosurgical Diagnosis—Chronic brain syndrome, posttraumatic

CASE No 31 H B, age 28 Examined Jan 26, 1954 Reg No 47563

History The hospital record on this case has been lost We have only the note on the consultation request from neurosurgery that it is a central lesion, and we have on our record the final diagnosis after our examination from neurosurgery

EXAMINATION

A *Hearing*—Within normal limits

B *Spontaneous Phenomena*—All within normal limits

C *Rotation*—To right	*To left*
Nystagmus—Hor to left *Amp*—Good, Dur 31 secs	Hor. to right *Amp*—Good, Dur 29 secs
Vert—Sub normal	Sub normal

HEAD 30° FORWARD

D *Caloric*—Right Ear	Left Ear
Nystagmus—Rot to left *Amp*—Fair, after 40 secs, Dur 80 secs	Rot to right *Amp*—Fair, after 40 secs, Dur 85 secs
Vert—Sub-normal	Subnormal

HEAD BACK

Nystagmus—Hor to left *Amp*—Good	Hor to right *Amp*—Good
Vert—Sub-normal	Subnormal
Atkinson Ratio—1 2	1 2 1

Summary—Responses to rotation slightly exaggerated especially from left tracts, responses to the caloric test are markedly depressed

Neurotologic Diagnosis—A high generalized lesion with early extension downward possible

Neurosurgical Diagnosis—Chronic brain syndrome

CASE No 50 R S, age 30 Admitted May 10, 1955 Examined June 17, 1955 Reg No 57290

History This right handed patient was rendered unconscious from a blow with a blunt weapon on the right parietal area on this date He was unconscious on admission, and X ray revealed a fracture of the right parietal bone On regaining consciousness he was totally aphasic with a partial flaccid paralysis of the right arm and leg

Examination—The paralysis and the aphasia are now much less, but his speech is rambling

A *Hearing*—A moderate perceptive deafness on the right, a 10 db to 20 db greater perceptive deafness on the left

B *Spontaneous Phenomena*—All within normal limits

HEAD 30° FORWARD.

Rotation—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Fair; Dur. 18 secs.	Hor. to right. *Amp.*—Fair; Dur. 16 secs.
Vert.—Fair; slight pallor and sweat.	Good; pallor and sweat.

HEAD 30° FORWARD.

Caloric—Right Ear.	Left Ear.
Nystagmus—None.	Rot. to right. *Amp.*—Finest possible; after 32 secs.; Dur. 58 secs.
Vert.—None.	None.

HEAD BACK.

Nystagmus—Hor. to right. *Amp.*—Good; irregular.	Obl. to right. *Amp.*—Large, irregular.
Vert.—Fair.	Fair.
Atkinson Ratio—...........	1:1.8.

Summary—Responses to rotation subnormal especially from right cts; responses to caloric tests absent from right vertical tracts, markly subnormal from left vertical tracts.

Neurotologic Diagnosis—Marked direct injury to upper brain stem ecially on right side with contrecoup injury to left cerebral and cerellar cortex.

Neurosurgical Diagnosis—Same.

CASE No. 44: N. C. J., age 34. Admission date March 20, 1955. Examed March 25, 1955. Reg. No. 56186.

History: This patient has been subject to grand mal attacks for several ars. He was sent to our department as a matter of interest in our dings.

EXAMINATION.

Hearing—Within normal limits.

Spontaneous Phenomena—Except a large vertical nystagmus on uprd gaze all are within normal limits.

HEAD 30° FORWARD.

Rotation—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 22 secs.	Hor. to right. *Amp.*—Good; Dur. 19 secs.
Vert.—Fair; no autonomic phenomena.	Fair; pallor, sweat and slight nausea.

HEAD 30° FORWARD.

Caloric—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Fair; after 35 secs.; Dur. 82 secs.	Rot. to right. *Amp.*—Good; after 20 secs.; Dur. 91 secs.
Vert.—Marked pallor and sweat.	Good.

HEAD BACK

Nystagmus—Hor to left *Amp*—Small	Hor to right *Amp*—Good
Vert—Fair	Good
Atkinson Ratio—1 2 3	1 4 5

Summary—Responses to rotation are just within normal limits caloric tests show marked depression in response from right tracts, the left being normal

Neurotologic Diagnosis—High localized lesion on the right

Neurologic Diagnosis—Grand mal with focus in the right temporal lobe

CASE No 39 E H, age 25 Admitted Feb 10 1952 Examined March 7 1952 Reg No 34020

History On January 18, 1951 this patient underwent brain surgery A medullo blastoma was removed subtotally from the right cerebellar hemisphere This was followed by X ray treatment with clinical improvement This case was sent to us for calibration of our tests

EXAMINATION

A *Hearing*—Within normal limits

B *Spontaneous Phenomena*—A vertical nystagmus on upward gaze Otherwise within normal limits

HEAD 30° FORWARD

C *Rotation*—To right	To left
Nystagmus—Hor to left *Amp*—Good, Dur 21 secs	Hor to right *Amp*—Good, with rapid decline, Dur 15 secs
Vert—None	None

HEAD 30° FORWARD

D *Caloric*—Right Ear	Left Ear
Nystagmus—Rot to left *Amp*—Fair, after 26 secs, Dur 107 secs	Rot to right *Amp*—Good, after 26 secs Dur 102 secs
Vert—Marked, pallor, sweat and emesis	Fair

HEAD BACK

Nystagmus—Hor to left *Amp*—Good	Hor to right *Amp*—Good
Vert—Good	Fair
Atkinson Ratio—1 4 1	1 3 9

Summary—Response to rotation, left canals normal, right canals depressed, response to caloric test, all canals respond normally

Neurotologic Diagnosis—Low lesion on right.

CASE No 40 W O'N, age 43 Admitted Feb 10, 1955 Examined Feb 18, 1955 Reg No 55500

History Staggering and stumbling to right and to left without vertigo for five years He is unable to walk or to stand in the dark No tinnitus or hearing loss

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Large; after 17 secs. Dur. 107 secs.	Rot. to right. *Amp.*—Large; After 15 secs. Dur. 84 secs.
Vert.—Marked; no autonomic phenomena.	Marked; no autonomic phenomena.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Large.	Hor. to right. *Amp.*—Large.
Vert.—Poor.	Poor.
Atkinson Ratio—1:6.3.	1:5.6.

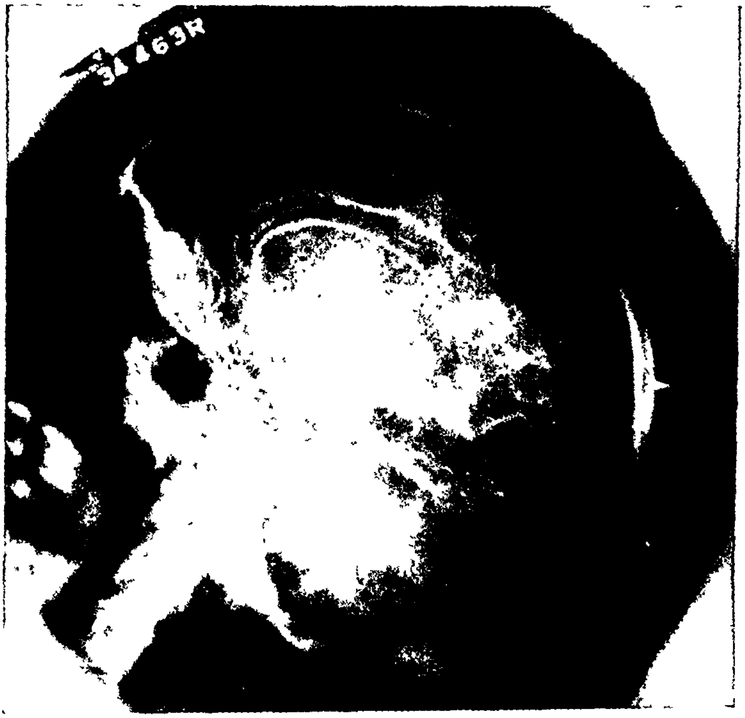

Fig. 6. Case No. 37. Arteriogram showing vascular displacement typical of hydrocephalus.

Summary—All nystagmus responses are markedly exaggerated; the vertigo in the horizontal tracts is abnormal to rotation; the nystagmus response from all tracts is hyperactive, whereas the vertigo is subnormal to the caloric test.

Neurotologic Diagnosis—Advanced lesion in the right cerebellum with extension upward, either infiltration or by encroachment on the foramina of Magendie and Luschka with beginning increase of intraventricular pressure

Neurosurgical Diagnosis—A central nervous system lesion probably cerebellar but exact site as yet undetermined

Subsequent History—On Dec 22 the optic discs became blurred, and the headache became much worse Arteriograms showed vascular displacement typical of hydrocephalus

On Dec 23 his condition became much worse with convulsions and unconsciousness Immediate surgery revealed an astrocytoma including the entire right cerebellar system which appeared to extend to the brain stem Marked hydrocephalus The patient expired on the table

CASE No 46 C W D age 39 Admitted April 25 1935 Examined May 6 1935 Reg No 57118

History This patient was sent from neurosurgery for calibration of our tests without previous history The only history we had was of staggering double vision headache and vomiting for several weeks progressive

EXAMINATION

Paralysis of the right VIth cranial nerve and marked slurring of speech

A *Hearing*—Within normal limits

B *Spontaneous Phenomena*—A second degree rotary nystagmus on forward gaze a marked vertical nystagmus on upward gaze and an oblique nystagmus up and to the left with the head in the back horizontal position There was marked staggering and deviation to the left on walking test Rombergism present to the right and backward in all three head positions The pelvic girdle reaction was positive being rigid as a board in attempts to tilt in all four directions

HEAD 30° FORWARD

C *Rotation*—To right	To left
Nystagmus—Hor to left *Amp*—Large Dur 31 secs swinging	Hor to right *Amp*—Large Dur 33 secs swinging
Vert—Good no autonomic phenomena	Good no autonomic phenomena

HEAD 120° FORWARD

Nystagmus—Rot to left *Amp*—Large Dur 19 secs	Rot to right *Amp*—Tiny Dur
Vert—Poor	Poor

HEAD 30° FORWARD

D *Caloric*—Right Ear	Left ear
Nystagmus—Rot to left *Amp*—Large after 15 secs swinging	Rot to right *Amp*—Large, after 25 secs swinging
Vert—Poor no autonomic phenomena	Poor no autonomic phenomena

HEAD BACK

Nystagmus—Hor to left *Amp*—Large swinging	Hor to right *Amp*—Large, swinging
Vert—Poor	Poor
Atkinson Ratio—1 8	1 58

Summary—1. The spontaneous and positional nystagmus indicates a central lesion; 2. The Romberg and pelvic girdle reactions tell of a cerebellar lesion; 3. The right sixth nerve paralysis denotes a high, right lesion; 4. The slurring speech is indicative of a high pontine lesion; 5. The marked exaggeration of all the nystagmus responses and the lessened to poor vertigo responses signifies primary low lesion, with upward spread by invasion or increase in pressure.

Neurotologic Diagnosis—Primary low (cerebellar) lesion with upward extension by invasion or increase of generalized pressure.

Neurosurgical Diagnosis—Information was now given that this patient had had a double mammectomy for bilateral adenocarcinoma a few months previously. The diagnosis then was metastasis to the cerebellum, with secondary higher metastases.

CASE NO. 38: L. L. R., age 33. Admitted Jan. 17, 1955. Examined Jan. 25, 1955. Reg. No. 54903.

History: This patient complained of persistent dizziness for two months, becoming worse two days previously; staggering, nausea and vomiting in spells lasting two to three days, continuous occipital headache and blurred vision for two weeks.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—A marked vertical nystagmus on upward gaze. Staggering and ataxia on walking test. He fell to the left with all positions of the head in the Romberg test, and his pelvis was rigid to tilt in all directions.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 42 secs.	Hor. to right. *Amp.*—Good; Dur. 42 secs.
Vert.—Poor; no autonomic phenomena.	Poor; no autonomic phenomena.

HEAD 120° FORWARD.

Nystagmus—Rot. to left. *Amp.*—Fair.; Dur. 22 secs.	Rot. to right. *Amp.*—Fair; Dur. 17 secs.
Vert.—Poor.	Poor.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Fair; After 22 secs.; Dur. 103 secs.	Rot. to right. *Amp.*—Fair; After 16 secs.; Dur. 114 secs.
Vert.—Fair; no autonomic phenomena.	Fair; no autonomic phenomena.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Fair.	Hor. to right. *Amp.*—Fair; very slow.
Vert.—Fair.	Fair.
Atkinson Ratio—1:4.6.	1:6.7.

Summary—Responses to rotation; marked exaggeration of nystagmus from horizontal tracts and depression of nystagmus from right vertical tract vertigo subnormal Responses to caloric tests, exaggeration of nystagmus from left verticals the other normal vertigo subnormal

Neurotologic Diagnosis—Posterior fossa (cerebellar) lesion expanding upward or increasing intraventricular pressure

Arteriogram—Anterior bowing and elevation of the right anterior cerebral artery to the right consistent with hydrocephalus

Neurosurgical Diagnosis—At operation on Jan 27, 1955, a glioma involving almost the entire left portion of the cerebellum and probably involving the brain stem

Case No 3 J R W age 30 Admitted Oct 25 1951 Examined Jan 11 1952 Reg No 40391

History This patient was admitted to the ophthalmologic department with ophthalmoplegia involving all three nerves bilaterally It was diagnosed Guillain Barre's syndrome, involving the eye muscle nuclei, cause unknown There was no benefit from the use of antibiotics and ACTH He was readmitted on Jan 9, 1952 for a check up There was improvement with only residual bilateral involvement of both lateral recti muscles It is believed that the disorder was of vascular origin

EXAMINATION

A *Hearing*—Within normal limits

B *Spontaneous Phenomena*—All within normal limits

Head 30° Forward

C *Rotation*—To right	To left
Nystagmus—Hor to left *Amp*—Good Dur 42 secs	Hor to right *Amp*—Good, Dur 26 secs
Vert—Good, little or no autonomic phenomena	Subnormal, slight nausea, no sweat

Head 30° Forward

D *Caloric*—Right Ear	Left Ear
Nystagmus—Rot to left *Amp*—Poor, After 32 secs , Dur 103 secs	Rot to right *Amp*—Poor, after 27 secs , Dur 94 secs
Vert—Good, nausea sweat and pallor	Good, nausea, sweat and pallor

Head Back.

Nystagmus—Hor to left *Amp*—Fair	Hor to right *Amp*—Good
Vert—Good	Good
Atkinson Ratio—1 3 2	1 3 4

Summary—Responses to rotation, right tracts normal, left tracts exaggeration Responses to caloric tests, bilateral depression

Neurotologic Diagnosis—Primary high central lesion with irritation downward on left

Case No 4 R J M, age 32 Admitted Dec 26, 1951 Examined Jan 1, 1952 Reg No 37713

History—This patient entered Hines Hospital in 1947 complaining of "black out" spells severe headache, nausea and vomiting At that time an ependymoma was subtotally removed from the fourth ventricle. He entered this hospital complaining of recurrent severe headache.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—A marked second degree horizontal nystagmus to the left, changing to vertical on upward gaze. He past pointed with both hands three inches to the left, staggered markedly on walking and fell backward with all positions of the head in the Romberg test; pelvis rigid.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Rot. to left. *Amp.*—Huge; Dur. 61 secs.	Rot. to right. *Amp.*—Large; Dur. 35 secs.
Vert.—Little or none; no autonomic phenomena.	Little or none; no autonomic phenomena.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Large; after 16 secs.; Dur. 103 secs.	Rot. to right. *Amp.*—Large; After 12 secs.; Dur. 119 secs.
Vert.—None.	None.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Huge.	Hor. to right. *Amp.*—Huge.
Vert.—None.	None.
Atkinson Ratio—1:6.4.	1:10.

Summary—The responses to both rotation and caloric tests are markedly exaggerated. Since we know the site of the original lesion we may trace its subsequent invasion, both upward and downward, with probable increase of pressure. On March 31, 1953, he was seen in the terminal stage, stuporous, incontinent and with left hemiplegia.

CASE NO. 14: W. L. M., age 28. Admitted Oct. 1, 1952. Examined Oct. 3, 1952. Reg. No. 38288.

History: In July, 1951, this patient noticed diplopia on looking to the left. This soon became permanent in all positions of gaze. In January, 1952, at biopsy a chondrosarcoma of the left sphenoid ridge was found. At the same time diabetes insipidus was diagnosed. In September, 1952, he developed blindness of the left eye with severe generalized left hemicrania and weakness of the right arm and leg.

EXAMINATION.

Paralysis left external rectus muscle.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

Head 30° Forward

C *Rotation*—To right	To left
Nystagmus—Hor to left *Amp*—Good, Dur 34 secs	Hor to right *Amp*—Good, Dur 40 secs
Vert—Subnormal, no autonomic phenomena	Subnormal, no autonomic phenomena

Head 30° Forward

D *Caloric*—Right Ear	Left Ear
Nystagmus—*Rot to left Amp*—Huge after 15 secs, Dur 110 secs	Rot to right *Amp*—Huge, After 16 secs, Dur 114 secs
Vert—Fair	Fair

Head Back

Nystagmus—Hor to left *Amp*—Huge	Hor to right *Amp*—Huge
Vert—Fair	Fair
Atkinson Ratio—1 73	1 71

Summary—The original site of this lesion is known to be high on the left Its progress upward and down to the posterior fossa can be traced in these tests Responses to rotation are exaggerated, especially from the right tracts, those from the caloric tests are most markedly exaggerated

Case No 19 T A P, age 28 Admitted Nov 5, 1952 Examined Nov 21 1952 Reg No 39077

History This is a case of grand mal of nine years' duration, with increasingly frequent typical generalized convulsions with aura

EXAMINATION

A *Hearing*—Within normal limits

B *Spontaneous Phenomena*—All within normal limits

Head 30° Forward

C *Rotation*—To right	To left
Nystagmus—Hor to left *Amp*—Huge Dur 45 secs	Hor to right *Amp*—Huge, Dur 37 secs
Vert—Fair, no autonomic phenomena	Fair, no autonomic phenomena

Head 30° Forward

D *Caloric*—Right Ear	Left Ear
Nystagmus—Rot to left *Amp*—Good, After 20 secs, Dur 114 secs	Rot to right Amp—Good, After 20 secs, Dur 113 secs
Vert—Poor, no autonomic phenomena	Poor, no autonomic phenomena

Head Back

Nystagmus—Hor to left *Amp*—Good	Hor to right *Amp*—Good
Vert—Poor	Poor
Atkinson Ratio—1 57	1 56

Summary Responses to both rotation and caloric tests show exaggeration Since the original site of this disturbance is known to be high, the spread of the irritation downward can be traced

CASE No. 29: G. A., age 59. Admitted Nov. 5, 1953. Examined Nov. 13, 1953. Reg. No. 46310.

History: The complaint in this case was frontal headache and failing vision for one year. On examination there was bilateral tubular vision only.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Rot. to left. *Amp.*—Huge; Dur. 32 secs.	Rot. to right. *Amp.*—Large; Dur. 32 secs.
Vert.—Subnormal.	Subnormal.

HEAD 120° FORWARD.

Nystagmus—Rot. to left. *Amp.*—Good; Dur. 15 secs.	Rot. to right. *Amp.*—Good; Dur. 19 secs.
Vert.—Fair; nausea.	Good; nausea and emesis.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Large; After 20 secs.; Dur. 160 secs.	Rot. to right. Amp.—Large; after 16 secs.; Dur. 158 secs.
Vert.—Marked nausea, sweat and emesis.	Marked nausea, pallor and emesis.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Huge.	Hor. to right. *Amp.*—Large.
Vert.—Good.	Good.
Atkinson Ratio—1:8.	1:8.3.

Summary—Response to rotation; moderate exaggeration; response to caloric tests; ; marked exaggeration.

Neurotologic Diagnosis—A high midline lesion expanding with increased pressure to posterior fossa.

Neurosurgical Diagnosis—On Nov. 16, 1953, a large sellar pituitary tumor was removed by right transfrontal craniotomy.

CASE No. 32: D. R., age 31. Admitted March 30, 1954. Examined April 23, 1954. Reg. No. 49020.

History: This patient presents diplopia; slight dizziness; ringing in the right ear, and low grade fever (99=100 F.) with sudden onset two weeks previously.

EXAMINATION.

Paralysis of the left rectus muscle. All other findings, both neurological and neurosurgical, were normal.

A. *Hearing*—The bone conduction and air conduction of both ears were approximately identical (all four curves) and were as follows: Dips to

10 db at 1 000 cps to 30 db at 2,000 cps, to 40 db at 4 000 cps, to 25 db at 4 000 cps, and to 35 db at 8,000 cps

B *Spontaneous Phenomena*—All within normal limits

HEAD 30° FORWARD

C *Rotation*—To right	To left
Nystagmus—Hor to left *Amp*—Huge, Dur 31 secs	Hor to right *Amp*—Huge, Dur 42 secs
Vert—Subnormal, no autonomic phenomena	Fair, no autonomic phenomena

HEAD 120° FORWARD

Nystagmus—Rot to left *Amp*—Fair, Dur 14 secs	Rot to right *Amp*—Large, Dur 22 secs
Vert—Marked slight sweat.	Marked, slight sweat

HEAD 30° FORWARD

D *Caloric*—Right Ear	Left Ear
Nystagmus—Rot to left *Amp*—Good After 28 secs, Dur 98 secs	Rot to right *Amp*—Large After 32 secs, Dur 128 secs
Vert—Good, marked pallor and sweat	Good pallor and sweat

HEAD BACK

Nystagmus—Hor to left *Amp*—Good	Hor to right *Amp*—Large
Vert—Good	Good
Atkinson Ratio—1 3 5	1 4

Summary—Marked exaggeration of responses of right tracts to rotation with less exaggeration from the left tracts, slight depression of the left tracts and normal response of the right tracts to caloric stimulation

Neurotologic Diagnosis—An expanding lesion high, involving the left abducens nucleus and the right vestibular nuclei, with extension by pressure or infiltration to posterior fossa or a disseminated lesion both high and low with first involvement high.

Medical Diagnosis—Multiple sclerosis

CASE No 43 J J age 34 Admitted March 16, 1955 Examined March 18 1955 Reg No 56193

History A few weeks ago this patient had a posterior fossa craniotomy at another hospital Prior to and since that time he has had trouble with walking failing vision, and lately some reduction in hearing acuity

EXAMINATION

A *Hearing*—A slight across the board reduction of bilateral perceptive type

B *Spontaneous Phenomena*—A large vertical nystagmus on upward gaze, past pointing six inches to the left with the left hand deviation to the right with ataxia and stumbling forward on walking Rombergism to the left with all positions of the head and rigidity of the pelvis on tilting

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Huge; Dur. 74 secs.	Hor. to right. *Amp.*—Large; Dur. 60 secs.
Vert.—Marked; no autonomic phenomena.	Marked—No autonomic phenomena.

HEAD 120° FORWARD.

Nystagmus—None.	Rot. to right. *Amp.*—Large; Dur. 20 secs.
Vert.—None.	Fair.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Large; after 20 secs.; Dur. 150 secs.	Rot. to right. *Amp.*—Large; After 15 secs.; Dur. 119 secs.
Vert.—None.	None.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Large.	Hor. to right. *Amp.*—Large.
Vert.—None.	None.
Atkinson Ratio—1:7.5.	1:8.

Summary—The spontaneous phenomena are those of a cerebellar lesion. All induced phenomena, both rotational and caloric, are exaggerated.

Neurotologic Diagnosis—The original posterior fossa lesion now shows extension upward, either from extension of the lesion or increase of the ventricular pressure.

Arteriogram—This shows a hemangioma of the posterior fossa with multiple dilated aneurisms of the entire circle of Willis.

CASE No. 47: E. D., age 59. Admitted May 4, 1955. Examined May 6, 1955. Reg. No. 57151.

History: This patient complained of difficulty with his vision, blurring and diplopia, and inability to look upward, with unsteadiness in walking. There was no nausea, headache or dizziness. The duration was about three weeks.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—A rotary nystagmus up and to the left in all directions of gaze except that upward gaze was impossible; he deviated markedly to the right on walking and on the Romberg test he fell backward when the head was turned to the left.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Large; Dur. 34 secs.	Hor. to right. *Amp.*—Large; Dur. 35 secs.
Vert.—Good; slight nausea.	Good; slight nausea.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Large; After 26 secs.; Dur. 125 secs.	Rot. to right. Amp.—Large; Afte 18 secs.; Dur. 133 secs.
Vert.—Fair.	Good; nausea and sweat.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Large.	Hor. to right. *Amp.*—Large.
Vert.—Fair.	Good
Atkinson Ratio—1:4.8.	1:7.4.

Summary—There is exaggeration of all responses to both rotation and caloric tests, showing irritation extending upward and downward from the tentorium, probably from a point just above the tentorium, as indi cated by his inability to gaze upward with both eyes.

Neurotologic Diagnosis—A lesion at or just above the tentorium, caus ing irritation both upward and downward.

Roentgenographic Studies—No deviation from the normal.

Neurosurgical Diagnosis—Damage to mesencephalon, locally destructive and not space occupying.

CASE No 27: S. H., age 37. Admitted Nov. 3, 1953 Examined Nov. 6, 1953. Reg No 45889.

History—No history was furnished us at the time of our examination except that the patient had a left side high tumor operated upon in 1945.

EXAMINATION.

A left moderate exophthalmos was apparent.

A *Hearing*—Within normal limits.

B *Spontaneous Phenomena*—A vertical nystagmus on upward gaze. Otherwise all within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 42 secs.	Hor. to right. *Amp.*—Good; Dur. 59 secs.
Vert—Moderate; no autonomic phenomena.	Moderate; no autonomic phenomena.

HEAD 120° FORWARD.

Nystagmus—Rot. to left. *Amp.*—Good; Dur. 20 secs.	Rot. to right. *Amp*—Good; Dur. 18 secs
Vert.—Fair.	Subnormal.

HEAD 30° FORWARD.

D *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Large; After 18 secs.; Dur. 129 secs	Rot. to right. *Amp.*—Large; After 17 secs.; Dur. 110 secs.
Vert.—Subnormal.	Subnormal.

Head Back.

Nystagmus—Hor. to left. *Amp.*—Large.	Hor. to right. *Amp.*—Large.
Vert.—Subnormal.	Subnormal.
Atkinson Ratio—1:7.1.	1:6.4.

Summary—All nystagmus responses to both rotation and the caloric tests are exaggerated. The vertigo responses are fairly normal. The latter finding indicates that the cerebellum is secondarily involved.

Neurotologic Diagnosis—A primary high lesion with secondary irritation of the posterior fossa structures.

Neurosurgical Diagnosis—Meningioma recurrent of left middle and frontal fossae, and left intraorbita. Generalized chronic brain syndrome associated with the neoplasm.

Case No. 11: M. W. H., age 34. Admitted April 29, 1952. Examined May 23, 1952. Reg. No. 35249.

History—This patient had acute meningo-encephalitis in 1946. Since then he has complained of severe head and neck pains, with a series of other vague complaints such as marked irritability, loss of libido, etc.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—There was consistent past-pointing to the left of two to three inches, Rombergism was present with sway to the left in all head positions; he was unable to complete the walking test because of ataxia and toppling to the left; the pelvis was rigid to the tilt test in all directions.

Head 30° Forward.

C. *Rotation—To right.*	*To left.*
Nystagmus—Hor. to left. *Amp.*—Large; Dur. 61 secs.	Hor. to right. *Amp.*—Large; Dur. 41 secs.
Vert.—Subnormal; no autonomic phenomena.	Subnormal; autonomic phenomena subnormal.

Head 120° Forward.

Nystagmus—Rot. to left. *Amp.*—Good; Dur. 30 secs.	Rot. to right. *Amp.*—Good; Dur. 25 secs.
Vert.—Fair.	Good; Nausea and emesis.

Head 30° Forward.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good; *After 20 secs.*; Dur. 121 secs.	Rot. to right. *Amp.*—Good; After 25 secs.; Dur. 120 secs.
Vert.—Fair.	Fair.

Head Back.

Nystagmus—Hor. to left. *Amp.*—Good.	*Hor. to right. Amp.*—Good.
Vert.—Fair.	Fair.
Atkinson Ratio—1:6.	1:4.8.

Summary—Both rotation and the caloric tests showed marked hyperirritability of both vestibular mechanisms, but the picture is irregular;

e g, the right side shows greater response to rotation than to the caloric tests, whereas the left side shows greater response to the caloric tests than to rotation The sense of vertigo is markedly subnormal, except that after rotation the right vertical canal mechanism showed hypersensitivity (Here the vestibular or canal mechanism includes both the end organ and the central tracts and connections)

Neurotologic Diagnosis—A widely disseminated lesion of an irritative nature in accord with residual effects of meningo encephalitis of the entire brain

Neurosurgical Diagnosis—Chronic brain syndrome (post encephalitic)

CASE No 12 W P, age 63 Admitted May 31, 1952 Examined July 11, 1952 Reg No 35921

History This patient complained of fainting spells, dizziness and tinnitus over the period of the past six or seven years His other symptoms and findings were typical of hypertension, the radial blood pressure being 260/104

EXAMINATION

A *Hearing*—The audiogram indicated a typical progressive senile perceptive deafness

B *Spontaneous Phenomena*—There was marked ataxia and staggering, Rombergism was present with falling to the left with all head positions, and the pelvis was rigid to the tilt test in all directions

HEAD 30° FORWARD

C *Rotation*—To right	To left.
Nystagmus—Hor to left *Amp*—Huge, Dur 21 secs	Hor to right *Amp*—Large Dur 25 secs
Vert—Fair, little or no autonomic phenomena	Marked with oscillations of the head and body, especially to right Emesis

HEAD 30° FORWARD

D *Caloric*—Right Ear	Left Ear
Nystagmus—Rot to left *Amp*—Good, After 40 secs, Dur 85 secs	Rot to right *Amp*—Large, After 25 secs, Dur 95 secs
Vert—Fair, slight nausea	Marked, nausea and emesis

HEAD BACK

Nystagmus—Hor to left *Amp*—Good	Hor to right *Amp*—Huge
Vert—Fair to good	Marked
Atkinson Ratio—1 2 1	1 3 8

Summary—The responses to rotation are within normal limits except for the bizarre oscillations of the head and body indicating cerebellar irritation The caloric responses are subnormal, indicating damage to the higher (brain stem and cerebral) tracts

Neurotologic Diagnosis—A generalized arteriosclerotic condition of the central tracts

On May 15, 1954, this patient expired in this hospital

Anatomic Diagnosis—Myocardial infarction, arteriosclerosis of the central nervous system

Case No. 17: B. R. M., age 32. Admitted Oct. 31, 1952. Examined Nov. 7, 1952. Reg. No. 38977.

History: The complaints were paraesthesia of the right little finger and of taste on the left side of the tongue; unsteadiness of gait with staggering, inability to tell just where his feet are in space with inability to make the left foot obey his will, and transient diplopia; all for about six months; no headache or convulsions.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—Staggered and deviated six feet to the right on walking test, and Rombergism present as a sway with falling to the right on turning the head to the right.

Head 30° Forward.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Huge; Dur. 63 secs.	Hor. to right. *Amp.*—Large; Dur. 55 secs.
Vert.—Subnormal.	Fair; slight pallor and sweat.

Head 30° Forward.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Large; After 25 secs.; Dur. 110 secs.	Rot. to right. *Amp.*—Good; After 20 secs.; Dur. 102 secs.
Vert.—Marked; nausea, sweat, pallor, emesis.	Marked; nausea and emesis.

Head Back.

Nystagmus—Hor to left. *Amp.*—Large.	Hor. to right. *Amp.*—Large.
Vert.—Marked.	Marked.
Atkinson Ratio—1:4.4.	1:5.1.

Summary—All responses to rotation and the caloric tests are exaggerated, the least being from the right vestibular system.

Neurotologic Diagnosis—A widely disseminated central disturbance.

Neurosurgical and Medical Diagnosis—Multiple sclerosis.

Case No. 22: W. P., age 33. Admitted March 12, 1953. Examined March 13, 1953. Reg. No. 42760.

History: There was a muscle weakness of the entire right side and a generalized numbness of two months' duration; it is now improving. Otherwise his history and general physical examination are negative.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—There was crossed past-pointing of both hands and persistent falling to the left with all positions of the head in the Romberg test.

Head 30° Forward

C *Rotation*—To right	To left
Nystagmus—Hor to left *Amp*—Good Dur 32 secs	Hor to right *Amp*—Good Dur 33 secs
Vert—Good, pallor, sweat and nausea	Good marked pallor, sweat and nausea

Head 30° Forward

D *Caloric*—Right Ear	Left Ear
Nystagmus—Rot. to left *Amp*—Good After 35 secs, Dur 98 secs	Rot to right *Amp*—Good, After 27 secs, Dur 107 secs
Vert—Good, pallor, sweat and emesis	Good pallor sweat and emesis

Head Back

Nystagmus—Hor to left *Amp*—Good	Hor to right *Amp*—Good
Vert—Good	Good
Atkinson Ratio—1 2 8	1 4

Summary—Responses of all canals to rotation are exaggerated, the right system is markedly depressed and the left system is normal to the caloric tests These findings together with the spontaneous phenomena, indicate generalized involvement with reversible damage high on the right

Neurotologic Diagnosis—A reversible (probably vascular) generalized disturbance most especially high on the right

Neurologic and Neurosurgical Diagnosis—A diffuse central lesion, probably vascular

Case No 24 B J B age 26 Admitted April 17, 1953 Examined May 1 1953 Reg No 42239

History For three years this patient had severe occipital headache with a swimming sensation most marked on coughing or sneezing

EXAMINATION

A *Hearing*—Within normal limits

B *Spontaneous Phenomena*—All within normal limits

Head 30° Forward

C *Rotation*—To right	To left
Nystagmus—Hor to left *Amp*—Good, Dur 40 secs	Hor to right *Amp*—Good, Dur 46 secs
Vert—Fair, profuse sweat	Fair, profuse sweat

Head 30° Forward

D *Caloric*—Right Ear	Left Ear
Nystagmus—Rot to left *Amp*—Good, After 22 secs, Dur 114 secs	Rot to right. *Amp*—Large, After 21 secs, Dur 119 secs
Vert—Little or none	Little or none

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Large.	Hor. to right. *Amp.*—Large.
Vert.—Little or none.	Little or none.
Atkinson Ratio—1:5.2.	1:5.7.

Summary—All responses to both rotation and the caloric tests are markedly exaggerated.

Neurotologic Diagnosis—A diffuse disturbance involving both the high and the low central nervous system.

Neurosurgical and Roentgenographic Diagnosis—Platybasia.

CASE No. 39: M. G., age 45. Admitted Dec. 28, 1954. Examined Jan. 28, 1955. Reg. No. 54475.

History: This patient complained of generalized pains in body and limbs (on previous admission to this hospital he had complained of various vague and undiagnosed body pains), inability to step up on curb, and staggering. A few months previously, on turning his head to the right there was a sudden pain as of an electric shock running up the right side of his head with flashing of red lights in front of his eyes, staggering and stumbling, but without true vertigo.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—Deviation and staggering to the right, Rombergism with falling to the right with all head positions and rigidity of the pelvis to tilting in all directions.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Large; Dur. 55 secs.	Hor. to right. *Amp.*—Good; Dur. 34 secs.
Vert.—Poor; no autonomic phenomena.	Poor; no autonomic phenomena.

HEAD 120° FORWARD.

Nystagmus—Rot. to left. *Amp.*—Large; Dur. 26 secs.	Rot. to right. *Amp.*—Good; Dur. 20 secs.
Vert.—Poor and perverted.	None.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good; After 20 secs.; Dur. 129 secs.	Rot. to right. *Amp.*—Good; After 11 secs.; Dur. 123 secs.
Vert.—None.	None.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Large; swinging.	Hor. to right. *Amp.*—Huge; swinging.
Vert.—None.	None.
Atkinson Ratio—1:6.4.	1:11.

Summary—All responses to both rotation and the caloric tests are markedly exaggerated, especially from the left system.

Neurotologic Diagnosis—A widespread disseminated disease of the central nervous system primary or most advancement in the cerebellum

Neurologic and Neurosurgical Diagnosis—Parenchymatous cortical cerebellar (and cerebral) atrophy.

Case No 42. L S H, age 43 Admitted March 1, 1955. Examined March 11, 1955. Reg. No 55878.

History: This case was sent to the otological department without a history for calibration of our tests. Our evaluation was wanted also.

EXAMINATION.

The patient was morose and introspective during the examination

A. *Hearing*—A mild across the board decrease of hearing acuity bilateral, especially on the left of perceptive type without recruitment or diplacusis.

B *Spontaneous Phenomena*—A deviation of seven feet on the walking test, Rombergism, with falling to the left with all head positions, and rigidity of the pelvis to tilting in all directions.

HEAD 30° FORWARD.

C *Rotation*—To right	To left.
Nystagmus—Hor. to left. *Amp*—Large, Dur. 19 secs.	Hor. to right. *Amp*—Large; Dur. 15 secs.
Vert—Fair, no autonomic phenomena	Fair; no autonomic phenomena.

HEAD 120° FORWARD

Nystagmus—Rot to left. *Amp*—Good; Dur. 17 secs.	Rot to right. *Amp*—Good; Dur. 15 secs
Vert—Fair.	Fair.

HEAD 30° FORWARD.

D *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp*—Tiny, After 43 secs.; Dur. 73 secs	Rot. to right. *Amp*—Small; After 37 secs.; Dur. 90 secs.
Vert—Little or none.	Little or none.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Very small.	Hor to right. *Amp.*—Good
Vert—Little or none.	Little or none.

EXAMINATION.

A. *Hearing*—A slight across-the-board lowering of the threshold for acuity of the perceptive type.

B. *Spontaneous Phenomena*—All within normal limits.

Head 30° Forward.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. Amp.—Fair; Dur. 14 secs.	Hor. to right. *Amp.*—Fair; Dur. 10 secs.
Vert.—Subnormal.	Subnormal.

Head 120° Forward.

Nystagmus—Rot. to left. *Amp.*—Good; Dur. 14 secs.	Rot. to right. *Amp.*—Fair; Dur. 11 secs.
Vert.—Marked; emesis.	Marked; emesis.

Head 30° Forward.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Small; After 35 secs.; Dur. 86 secs.	Rot. to right. *Amp.*—Small; After 37 secs.; Dur. 86 secs.
Vert.—Subnormal; emesis.	Subnormal; emesis.

Head Back.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Large.
Vert.—Fair; emesis.	*Fair; emesis.*
Atkinson Ratio—1:2.4.	1:2.3.

Summary—All responses to rotation and to the caloric tests are markedly subnormal except those from the left vertical canal system.

Neurotologic Diagnosis—A disturbance (degenerative?) of the entire central nervous system—both high and low.

Neurosurgical Diagnosis—Tic douloureaux. No other pathology found.

Case No. 13: D. E. W., age 21. Admitted Sept. 14, 1952. Examined Sept. 21, 1952. Reg. No. 35818.

History: This patient complained of uncontrollable sleeping. He was courtmartialed twice while in service for sleeping on post. He now falls asleep while at work. At one time he slept continuously for 43 hours. This was sleep and not coma. True narcolepsy has been ruled out.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

Head 30° Forward.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 24 secs.	Hor. to right. *Amp.*—Good; *Dur.* 21 secs.
Vert.—Marked; pallor and profuse sweat.	Marked; pallor and profuse sweat.

HEAD 30° FORWARD

D *Caloric*—Right Ear	Left Ear
Nystagmus—Rot to left *Amp*—Poor After 56 secs, Dur 76 secs	Rot to right *Amp*—Poor, After 55 secs, Dur 65 secs
Vert—Marked, pallor, nausea and sweat	Marked, pallor, nausea and sweat

HEAD BACK

Nystagmus—Hor to left *Amp*—Fair	Hor to right *Amp*—Fair
Vert—Good	Good
Atkinson Ratio—1 1 4	1 1 2

Summary—Responses to rotation normal, responses to caloric tests markedly subnormal

Neurotologic Diagnosis—A high diffuse lesion

Neurosurgical and Neuropsychiatric Diagnosis—Conversion hysteria

CASE No 15 G T M, age 52 Admitted Sept 30, 1952 Examined Oct 3, 1952 Reg No 38344

History For the past two and one half years this patient had recurrent attacks of dizziness, lightheadedness, nausea and diplopia when under stress

EXAMINATION

A *Hearing*—Within normal limits

B *Spontaneous Phenomena*—Except stiffness of his pelvis to the tilting tests all were within normal limits

HEAD 30° FORWARD

C *Rotation*—To right	To left
Nystagmus—Hor to left *Amp*—Huge, Dur *33 secs*	Hor to right *Amp*—Good, Dur *26 secs*
Vert—Marked (fell to right during the turning) pallor and nausea	Good (slumped in chair during the turning), pallor and nausea

HEAD 30° FORWARD

D *Caloric*—Right Ear	Left Ear
Nystagmus—Rot to left *Amp*—Good, After 28 secs, Dur 95 secs	Rot to right *Amp*—Good, After 22 secs, Dur 91 secs
Vert—Good, emesis	Good, emesis

HEAD BACK

Nystagmus—Hor to left *Amp*—Good	Hor to right *Amp*—Good
Vert—Good.	Good
Atkinson Ratio—1 3 4	1 4 1

Summary Responses to rotation are exaggerated especially from the left system, responses to the caloric tests are nearly normal, being only slightly reduced from the right system

Neurotologic Diagnosis—A midline cerebellar lesion, more to the left hemisphere, and with early secondary effects on the higher nervous system.

Neurosurgical and Neuropsychiatric Diagnosis—Shortly after the neurotologic examination and while still hospitalized this patient developed a typical paranoid schizophrenic syndrome and so diagnosed.

Case No. 21: W. C., age 47. Admitted Dec. 19, 1952. Examined Dec. 26, 1952. Reg. No. 39907.

History: This patient has a long history of hospitalizations for vagotomy, subtotal gastrectomy, and for closure of an oro-antral fistula with severe pain in his left cheek. To control pain he requires codein grs. ss every three hours. There may be drug addiction. He has glycosuria of unknown origin. His left eye turns in, which he states followed an injection of his left sphenopalatine ganglion with phenol, which was done recently in an effort to control his pain.

EXAMINATION.

There is a marked weakness, almost a paralysis, of the left external rectus muscle.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

Head 30° Forward.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 22 secs.	Hor. to right. *Amp.*—Good; Dur. 20 secs.
Vert.—Subnormal.	Subnormal.

Head 30° Forward.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good; After 45 secs.; Dur. 72 secs.	Rot. to right. *Amp.*—Good; After 38 secs.; Dur. 92 secs.
Vert.—Poor.	Poor.

Head Back.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Good.
Vert.—None.	None.
Atkinson Ratio—1:1.6.	1:2.4.

Summary—Responses to rotation are normal; those from caloric testing are markedly subnormal.

Neurotologic Diagnosis—A diffuse or disseminated high lesion.

Neurosurgical and Neurologic Diagnosis—No evidence of neuropathy found.

Case No. 23: W. P. B., age 28. Admitted March 19, 1953. Examined April 10, 1953. Reg. No. 41637.

History—This patient complained of frontal headache and mild convulsive seizures. One year ago he had a carotid ligation to reduce a possible intracranial hemangioma as there is an extensive hemangioma involving the face and bones (frontal) of the skull. He is a chronic alcoholic.

EXAMINATION

A *Hearing*—A dip to 35 db in each ear at 4 000 cps Otherwise within normal limits

B *Spontaneous Phenomena*—All within normal limits

HEAD 30° FORWARD

C *Rotation*—To right	To left
Nystagmus—Hor to left *Amp*—Good *Dur 24 secs*	Hor to right *Amp*—Good, Dur 22 *secs*
Vert—Fair to good	Fair to good

HEAD 30° FORWARD

D *Caloric*—Right Ear	Left Ear
Nystagmus—Rot to left *Amp*—Fair, After 37 secs Dur 85 secs	Rot to right *Amp*—Good After 41 secs Dur 74 secs
Vert—Fair to good, nausea and sweat	Good nausea and sweat

HEAD BACK

Nystagmus—Hor to left *Amp*—Fair	Hor to right *Amp*—Good
Vert—Fair	Good
Atkinson Ratio—1 2 3	1 1 7

Summary—Responses to rotation are normal, to caloric tests they are markedly subnormal

Neurotologic Diagnosis—A probable high organic lesion

Neurosurgical Diagnosis—There is no evidence of an intracranial growth at this time

CASE No 36 A B A, age 45 Admitted Nov 2, 1954 Examined Nov 5 1954 Reg No 53422

History This patient complained of severe headache of three months' duration Three nights ago he awakened from sleep dizzy and suffocating He arose from his bed, and on attempting to walk fell backward to the ground On arising he had severe vertigo, nausea and staggering all of which are persistent One week before admittance he had a black out while at work One year ago he was struck on the head with an iron beam

EXAMINATION

A *Hearing*—Within normal limits

B *Spontaneous Phenomena*—Marked staggering, the Romberg test showed swaying with forward gaze and backward falling with the head to right or left, and the pelvis was rigid to tilting in all directions

HEAD 30° FORWARD

C *Rotation*—To right	To left
Nystagmus—Hor to left *Amp*—Huge Dur 41 secs	Hor to right *Amp*—Large, Dur 36 secs
Vert—Good, autonomic phenomena subnormal	Good, autonomic phenomena subnormal

HEAD 120° FORWARD.

Nystagmus—Rot. to left. *Amp.*—Large; Dur. 20 secs.	Rot. to right. *Amp.*—Good; Dur. 23 secs.
Vert.—Fair; slight pallor and sweat.	Fair to good; pallor, sweat, nausea.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good; After 25 secs.; Dur. 103 secs.	Rot. to right. *Amp.*—Good; After 21 secs.; Dur. 98 secs.
Vert.—Good; slight pallor, sweat, nausea.	Good; pallor, sweat, nausea, emesis.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Good.
Vert.—Good.	Good.
Atkinson Ratio—1:4.	1:4.6.

Summary—Responses to rotation are exaggerated; to caloric tests they are normal.

Neurotologic Diagnosis—A lesion of the midline structures of the cerebellum (probably vascular in origin).

Neurosurgical and Neurophychiatric Diagnosis—Hysteria.

CASE No. 41: E. R. M., age 31. Admitted Feb. 21, 1955. Examined Feb. 25, 1955. Reg. No. 55719.

History—In March, 1952, there was fogging and then blindness of the left eye which lasted three weeks, and recovered spontaneously with no known cause. Three months later he noticed tightness of the head with irregular staggering and falling; again with recovery in three weeks. Three weeks ago he suffered attacks of loss of control of the left leg, and he overshot or undershot the mark with his foot, on attempting to step up on a curb, and his left leg would not track with his right leg on making a turn. There was no history of pain or head injury.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Fair; Dur. 38 secs.	Hor. to right. *Amp.*—Good; Dur. 40 secs.
Vert.—Poor; no autonomic phenomena.	Poor; no autonomic phenomena.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good; After 24 secs.	Rot. to right. *Amp.*—Good; After 23 secs.
Vert.—Poor; no autonomic phenomena.	Poor; no autonomic phenomena.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Good.
Vert.—Poor.	Poor.
Atkinson Ratio—1:4.3.	1:4.7.

Summary—To rotation; nystagmus exaggerated, vertigo poor; to caloric tests; nystagmus at upper limit of normal, vertigo poor.

Neurotologic Diagnosis—An early lesion (probably vascular) of the cerebellum, midline.

Neurosurgical and Medical Diagnosis—No evidence of neoplasm. No diagnosis made.

CASE No. 20: E. J. G., age 47. Admitted Nov. 12, 1952. Examined Nov. 21, 1952. Reg. No. 39321.

CASE No. 48: B. L. G., age 41. Admitted May 9, 1955. Examined May 6, 1955. First examined June 5, 1953. Reg. No. 57253.

CASE No. 51: G. G., age 56. Admitted June 9, 1955. Examined June 10, 1955. Reg. No. 57879.

These three cases are not being reported in detail because they were lesions of the cerebello-pontine angle, and as such presented their classical syndrome of a complete peripheral lesion on the side of the lesion with blocking of the tracts from the vertical canals on the opposite side. This syndrome, being so diagnostic of the angle lesion, which seems to present the picture of neither a high nor a low lesion, removes these cases from the clinical problem considered by the present work. For this reason these cases are removed from the statistical problem also. This limits the number of cases to a total of 49, rather than the actual total of 52.

SUMMARY OF CASE REPORTS.

Certain presumptions may be made reasonably from the evidence presented by these clinical observations. These presumptions are:

1. The tentorium appears to be the geographic dividing line between high and low lying lesions as here presented.

2. When a lesion is high and localized and the equilibratory system is affected, the responses to the cold caloric test are affected, whereas the responses to rotation remain within normal limits.

3. When a lesion is low and localized the reverse syndrome will appear. The responses to rotation are disturbed, and the responses to the cold caloric test remain within normal limits.

4. In either of the above conditions, if the lesion is irritative the responses will indicate hyperactivity; if the lesion is destructive the responses will indicate hypoactivity.

5. As the effects of the lesion spread upward or downward the syndrome changes accordingly. This is quite evident in the case of a low lying lesion producing a block to normal ventricular drainage.

6. A generalized lesion is indicated by the presence of affected responses, both to rotation and to the cold caloric test.

7. At the present writing there must be corroborating neurological or neurosurgical evidence before surgery is entertained, although the neurosurgeon may well be alerted to an impending surgical crisis.

8. The response to either rotation or caloric stimulation that seems to be basically affected is nystagmus. Effects on vertigo, falling or the autonomic phenomena seem to be secondary.

9. A lesion limited to the cerebello-pontine angle seems to be neutral so far as the differentiation considered here is concerned, seeming to have its own syndrome. For this reason the three cases recorded are not considered statistically.

10. On the basis of the observations made here correct localizations were made in 42 of the 49 cases or in 85.7 per cent of the cases observed.

11. As supportive evidence of the clinical observations reported here we have considered a series of cases reported elsewhere.[7] In their text Drs. Jones and Fisher report a series of cases to illustrate the localization of peripheral and central lesions. The pattern considered in this paper was not considered by them. A series of 22 intracranial lesions is reported. These are cases numbered 11 to 32 inclusive. Cases 26 to 32 are angle lesions and so may be excluded. Of the remaining cases one was undiagnosed, leaving 14 cases for consideration. Of these 14 cases, 10 cases (numbered 11, 12, 13, 15, 16, 19, 20, 21, 23 and 25) or 71.4 per cent show the high-low pattern unmistakably.

ANATOMICO-PHYSIOLOGIC CONSIDERATIONS.

In attempting to understand and to explain the evidence presented by the cases reported the following questions are encountered:

1. Do high lying (upper rhombencephalic, mesencephalic, diencephalic or even telencephalic) centers exist for the mediation of equilibratory phenomena, most particularly for nystagmus?

2. Do low lying posterior fossa or cerebellar equilibratory centers having to do with nystagmus exist?

3. Is there any basic difference in the mechanisms of response to rotation and to the cold caloric test?

4. If the answers to these questions are affirmative, can a theoretical mechanism explaining the modus operandi of the high and low syndromes be established?

Let us consider these problems.

Since Breuer[3] in 1875 noted that stimulation of the labyrinth of the pigeon produced eye movements, and Barany[4,5] in 1906 made his classic observations that caloric stimulation of the labyrinth in humans produced true nystagmus, and that this nystagmus lasted longer than the duration of the peripheral stimulus, the existence of connections between the labyrinth and the eye muscle nuclei via the structures of the brain stem has been recognized. The literature since then is so replete with experimental and clinical data proving their existence as to preclude the possibility of presenting a complete bibliography of this phase of the problem in a discussion of this type. It must suffice to state that the anatomic foundation of the reflex arc in question was laid by Ramon y Cajal between 1887 and 1892 and finally reported by him in 1893.[6]

The later development of this phase of the question may be traced by noting the simplicity of the mechanism as conceived by Isaac Jones[7] in 1918 and comparing it to the mechanism for the eye muscle reflexes as conceived by Lorente de No[8,9,10] in which the direction, the amplitude and the duration of these reflex responses (most especially nystagmus and compensation after unilateral labyrinthine destruction with which we are concerned here) are made a function of the substantia reticulata, a structure that forms one of the pathways between the labyrinth and the eye muscle nuclei. These various pathways have been accepted by anatomists, physiologists and clinicians alike. Lesions involving these tracts to and through the mid-brain, and their connections with the cerebral cortex, the thalamus, the hypo-thalamus and the extra-pyramidal nuclei, are known to have their clinical effects on the process of equilibration.[11] The eye muscle-labyrinthine reflexes (postural and nystagmatic) are most importantly involved in the considerations of this discussion. These structures and pathways are involved, whether the labyrinth be stimulated by rotation or by caloric excitation.[8,9]

The existence of cerebellar centers governing or mediating nystagmus has not been established for as long a time. Such centers were suspected (*e.g.*, Bauer and Leidler[12] noted that the duration of nystagmus was prolonged by a lesion in the vermis), but not until 1921 were they proven experimentally in the rabbit by Hoshino.[13] He noted that tracts connecting the vestibular nerves with the cerebellum had been recognized by "Bechterew, van Gehuchten, Andre Thomas, Edinger, Winkler, Ramon y Cajal, Fuse, etc.," but he felt that any concept of a connection between eye motion and the cerebellum was vague. He stimulated the cerebellum with a Faradic current and noted that with the head in a horizontal position eye deviation occurred to the stimulated side. After bilateral destruction of the labyrinths the same effect was obtained. He also found that stimulation of the vermis increased the intensity of both post rotatory and calorically induced nystagmus if the labyrinths were intact.

In 1951 Cranmer[14] reported a series of experiments working with monkeys. After observing the effects of both stimulation and destruction of the superior and lateral vestibular nuclei, he noted that Faradization of the right side of the pyramis of the cerebellum produced slow right conjugate deviation (left nystagmus) of the eyes. Stimulation of the left side of the pyramis caused left conjugate deviation (right nystagmus) of the eyes. Destruction of the pyramis on each side reversed these phenomena. The right nucleus fastigii was then stimulated and a left slow conjugate deviation (right nystagmus) of the eyes was noted. Thus he demonstrated an inhibitory action of the pyramis over the nucleus fastigii, which in its turn has an inhibitory action on the vestibular nuclei of the opposite side of the pons.

Mygind in 1953[15] stated that in unilateral labyrinthine destruction, after compensation has taken place, the cerebellum is no longer an inhibitor only. It also becomes an activator as necessity demands.

It should be noted that both Cranmer and Mygind used only position and rotation to effect the alterations in deviation and nystagmus. No caloric tests were mentioned.

Crosby in 1953[16] confirmed the thesis that the nucleus fastigii and the vermis are mediators of nystagmus.

Cawthorn in 1954[17] was of the opinion that when positional nystagmus has a central origin that site is in the midline of the cerebellum (Table I of his paper); however, he notes that a caloric abnormality in a surprisingly high percentage of such cases exists (Table VI), which would seem to devaluate my own observations.

Thus we conclude that there are centers mediating nytagmus in the cerebellum (subtentorial) as well as in the higher (supratentorial) areas.

In 1931 Lorente de No[8] described for the first time the fact that each face of the cristae of the canals, and the entire macule of the utricle are supplied by three types of nerve fibers; that each type has a regional distribution in the cristae and in the macula and that each type ends in a specific type of end-organ (see Figs. 7 and 8). These structures of the cristae and the utricular macula bear a striking resemblance to each other.

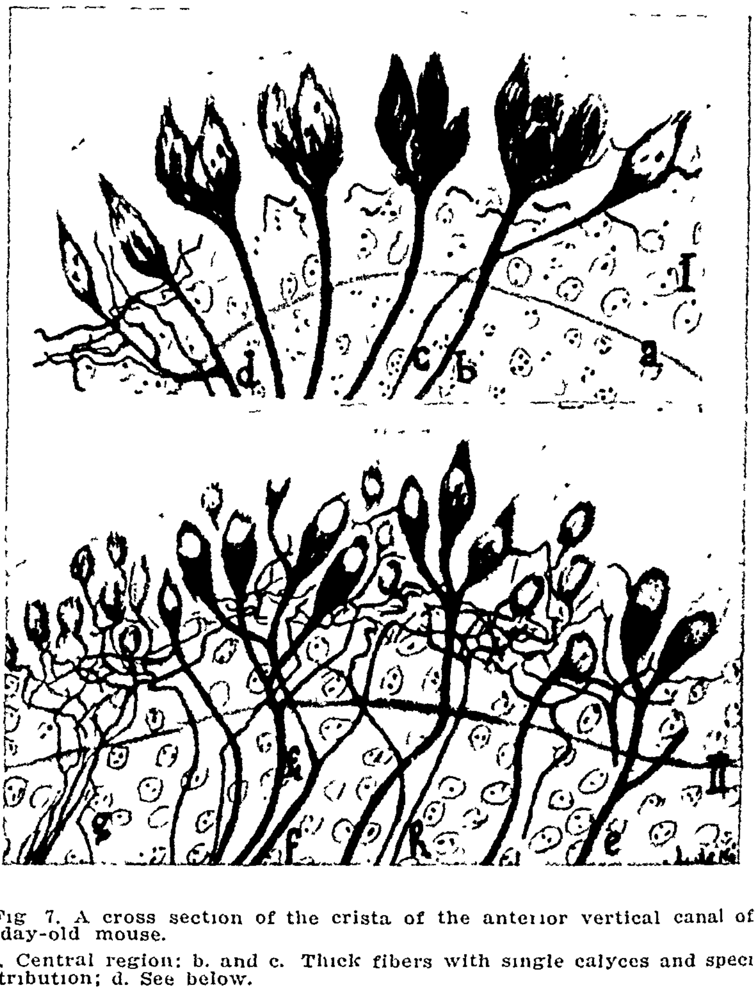

Fig 7. A cross section of the anterior vertical canal of a 25-day-old mouse.

I. Central region: b. and c. Thick fibers with single calyces and specific distribution; d. See below.

II. Peripheral region: d., e. and f. Medium thick fibers with branching and multiple calyces showing overlapping distribution; g. and h. Thin fibrils forming an intraepithelial plexus ending in hull-like coating for the hair cells.

The macule of the saccule also contains three somewhat similar but much less differentiated fibers and end-organs (see Fig. 9); furthermore each of the three types of nerve fibers maintains a regional or trunk line grouping within the vestibular nerve, and this specific grouping is maintained by the parent cells within the ganglion of Scarpa.

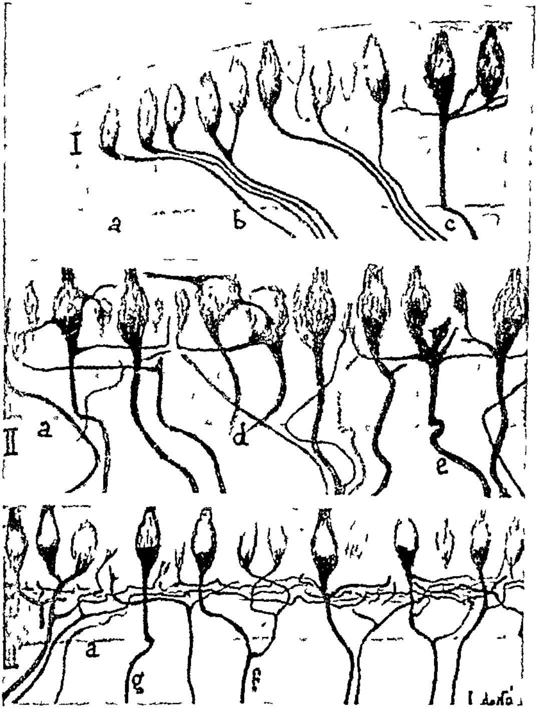

Fig 8 Cross section of the utricular macula of a 25-day-old mouse I Central region, II Intermediate region, III Peripheral region

Note the similarity of the fibers and their endings to those of crista in Fig 7

The specificity of these structures surely indicates that each type responds to a specific type of stimulus.

In the same communication Lorente de No points out that the cristae have three possible methods of stimulation. These are:

1. Displacement of the hair cells by displacement of the cupola from momentary current action of the endolymph.

2. Distortion of the cristae by distortive action of currents of the perilymph acting on the enclosed membranous labyrinth.

3. The action of centrifugal force producing outward pull and displacement.

He feels that stimulation of the hair cells of the maculae is produced by alteration in shape of the overlying otolith membrane whose structure varies with the type of end-cells in the macula on which it rests. This process is a comparatively slow one when compared with the instant response of the cristae to sudden motion. It may well account for the time lag of postural eye position changes that occurs after experimental ablation of the canal responses. This alteration in shape of the otolith membrane may be produced by gravity or by centrifugal force.

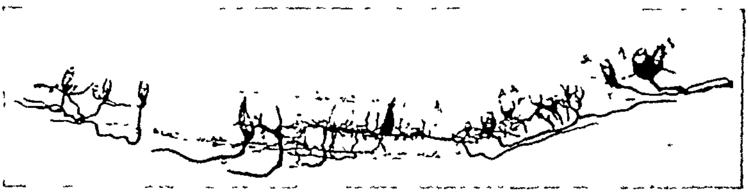

Fig. 9. Cross section of the saccular macula of the rat. Note on comparison with the structure of the utricular macula in Fig. 8 the markedly lessened degree of differentiation of the types of fibers, the end cells and their distribution.

It is evident at once that rotation brings into action all three modalities—hair cell displacement by endolymph deformation of the cristae, distortion of the membranous labyrinth by perilymph currents, and the action of centrifugal force.

It is equally evident that in the caloric tests centrifugal force can play no part. There is then an essential difference in the mechanics of the two processes. There is also the strong probability that the rotational tests as they are performed clinically with high acceleration and long continued duration may bring into play the maculae, especially the utricular maculae.

Since the answers to the first three of the questions encountered are affirmative, may we assume hypothetically that impulses resulting from centrifugal force have a particular route to the cerebellum, most especially to the pyramis and to the fastigial nucleus; that when a low lying lesion affects these areas of the cerebellum the rotational tests are affected, whereas the caloric tests that do not invoke centrifugal force are unaffected; that per contra when a lesion lying higher (perhaps above the tentorium) does not affect these cerebellar areas the added influence of centrifugal force may allow nystagmus to appear with little or no clinical evidence of alteration of the results of the rotational tests, whereas the caloric tests without this added influence are clinically disturbed.

These are the observations and the deductions of a clinical otologist who is interested in neurotology. It will remain for an experimental scientist in these fields to substantiate or nullify the deductions. The observed clinical phenomena, being factual, should stand while a more nearly correct explanation is sought should the deductions be disproven.

SUMMARY.

1. A syndrome evidenced by high central disturbances and a syndrome evidenced by low lying disturbances of the central nervous system have been described.

2. Their use in differential diagnosis has been indicated.

3. A hypothetical but entirely possible anatomico-physiologic explanation for these syndromes has been presented.

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THE SURGICAL ANATOMY OF THE ANTERO-CAUDAL PORTION OF THE NASAL SEPTUM: A STUDY OF THE AREA OF THE PREMAXILLA.*

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INTRODUCTION.

It is the purpose of this paper to explore the anatomy of the caudal end of the nasal septum with the object of gaining a better understanding of the surgical anatomy. In performing septal surgery, a detailed knowledge of the framework of the premaxillary area is essential. A surgical procedure upon the septum, that requires a thorough understanding of this structure will be presented.

The desired information about the anatomy of the premaxilla and septal operative technique is not available in a single publication. It would seem advantageous to assemble this information of the surgical anatomy of the antero-caudal end of the nasal septum into one dissertation.

The author wishes to express his appreciation for the important groundwork laid for this study while attending seminars presented by Dr. M. H. Cottle.[1]

OBJECTIVES.

A detailed study of cadaver dissections and a report of observations made upon many surgical procedures on the antero-caudal end of the nasal septum will be presented. This is necessary in order to justify an improved operative procedure.

The objectives are:

1. To present a detailed description of the mucoperichon-

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drial and mucoperiosteal reflections of the antero-caudal end of the nasal septum.

2. To assemble pertinent data derived from a study of the rigid structures from this same area on cadavers and operated patients.

3. To emphasize the failure of the standard submucous resection incision to provide clear access to the premaxillary area.

4. To investigate the concept that an intranasal incision and "undermining the base of the nose"[2] provides an improved exposure for correcting caudal basal spurs and deviations of the nasal septum.

NOMENCLATURE.

1. Antero-Caudal End of the Nasal Septum: This is divided into three parts; bony, cartilaginous, and membranous septum. The bony portion of the septum is composed of a part of the premaxilla and its process (anterior nasal spine and premaxillary wing), the crest of the superior maxilla articulating superiorly with the vomer and anteriorly with the premaxillary wing, and the vomer having attachments with the premaxillary wing. This bony portion of the antero-caudal end of the nasal septum will be designated the premaxillary area in this dissertation (see Fig. 1).

The anterior nasal spine is a sharp median spur of bone that projects from the lower margin of the pyriform aperture, and its base is formed by the two maxillae meeting in the midline. Projecting from its base in the midline is a sharp prominence, the median ridge, which extends from the alveolar arch and terminates in the point of the anterior nasal spine.*

The premaxillary wing is posterior to the anterior nasal spine and contiguous with it. The wing projects upward from the midline of the upper face of the floor of the pyriform aperture.

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* Embryologically the point of the anterior nasal spine is derived from the two premaxillary bones. The median ridge and base of the spine comes from the two maxillary bones.

The cartilaginous portion is formed by the cartilage of the septum that is attached inferiorly to the anterior nasal spine, to the premaxillary wing, and to the anterior part of the vomer

The membranous septum is made up by the juxtaposition of two mucocutaneous flaps The layers of the membranous septum extend forward from the free anterior margin of the septal cartilage to the cutaneous coverings of the medial crurae of the lower lateral cartilages, which form the cartilaginous support of the columella

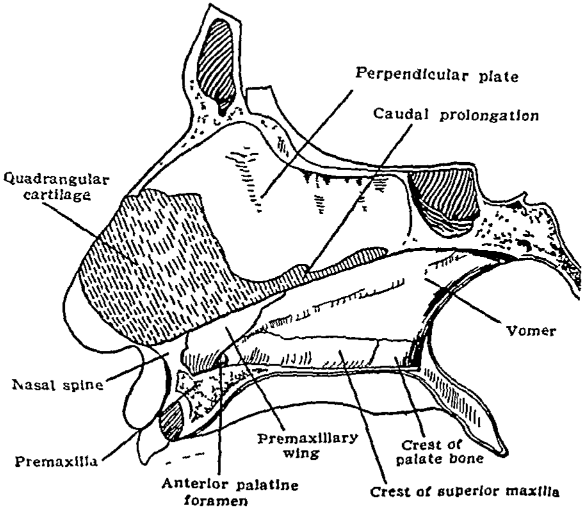


Fig 1 The Antero Caudal End of the Nasal Septum (modified from Mosher[4])

2 The Adult Septum Anatomical terms that name the structures in the various diagrams are used interchangeably *a.* Bony Portion: The premaxillary wing (Mosher[4]) has the same meaning as the incisive crest (Freer[5]). The terms crista nasalis maxillae (Loeb[3]), crest of superior maxilla (Mosher[4]), and palatal process of superior maxilla (Lothrop[6]) are synonymous.

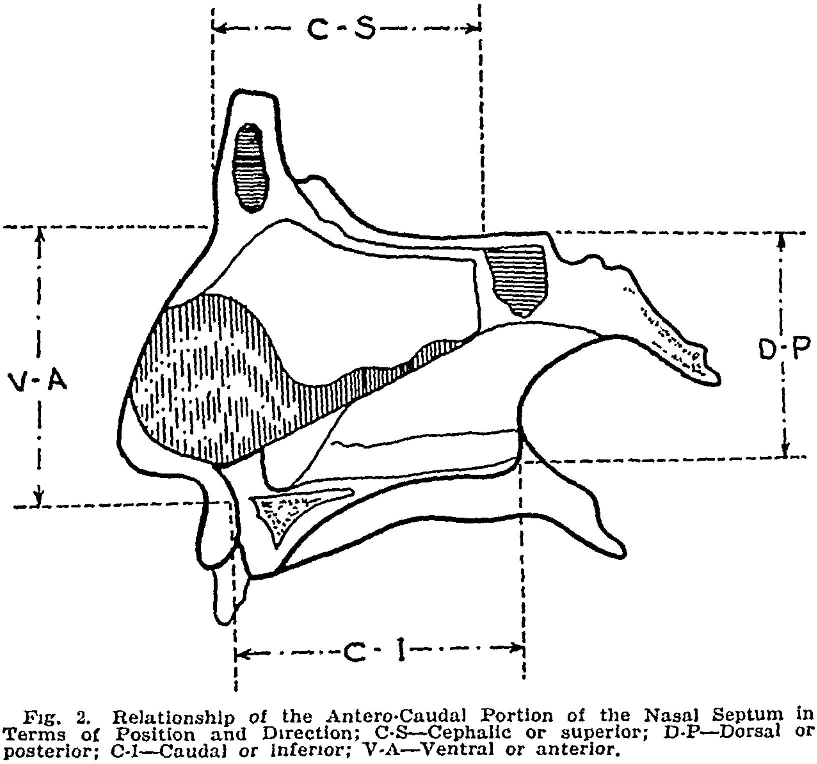


Fig. 2. Relationship of the Antero-Caudal Portion of the Nasal Septum in Terms of Position and Direction; C-S—Cephalic or superior; D-P—Dorsal or posterior; C-I—Caudal or inferior; V-A—Ventral or anterior.

b. Cartilaginous Portion: The terms quadrilateral cartilage, quadrangular cartilage (Mosher[4]), and cartilage septi nasi (Loeb[3]) are synonymous.

c. Membranous Septum: The membranous septum (Loeb,[3] Converse[7]) has the same meaning as the septum mobile nasi (Loeb[3]).

3. Terms of Position and Direction (see Fig. 2): The three fundamental planes of the body are the sagittal, the transerse, and the frontal. The vertical plane through the longi-udinal axis of the trunk, dividing the body into right and left alves is the median or midsagittal plane, and any plane paral-l to this is a sagittal plane. Any vertical plane at right angles o a sagittal plane, and dividing the body into front and rear ortions, is a frontal (coronal) plane. A plane across the body t right angles to sagittal and coronal planes is a transverse or orizontal plane.

Terms pertaining to the front of the body are anterior or entral; to the rear, posterior or dorsal; upper is designated as uperior or cephalic; and lower as inferior or caudal. The rm medial means nearer the midsagittal plane, and lateral, urther from that plane.

It should also be noted that the terms ventral, dorsal ephalic, and caudal are independent of the body posture, and, herefore, apply equally well to corresponding surfaces of ertebrates in general with horizontal body axis.[8]

The anterior free end of the septum (quadrilateral carti-age) refers to the free margin of this cartilage in continuity ith the membranous septum. The caudal bony portion of the asal septum refers to the area of the premaxilla.

In this thesis, the parts of the premaxilla pertinent to the audal border of the nasal septum are the anterior nasal spine nd premaxillary wings. The remainder of the premaxilla, as escribed by Ashley-Montagu,[9] enters into the structure of the yriform aperture and the anterior hard palate.

4. Nares: The nares is bounded below by an internal and xternal naris between which lies the floor of the nasal vesti-ule. The inferior part of the internal narial fold extends long the crest of the pyriform aperture.[7]

5. Pyriform Aperture. This aperture is the pear-shaped nterior external opening in the bony skeleton that bounds oth nasal fossae. Its lateral margin is called the ascending or ateral edge. The floor of the pyriform aperture varies [illegible] ge and with the ethnic origin of the individual.[10] In [illegible] aces the adult floor of the pyriform aperture has [illegible] fined edge anteriorly; the crest of the pyriform aperture. This crest is noticeably deficient in the negro race and, therefore, the floor of the pyriform aperture is continuous with and on the same horizontal plane as the floor of the nose.

REVIEW OF LITERATURE.

a. Historical: The voluminous literature relevant to the nasal septum was reviewed, and a certain pattern of thought concerning the submucous resection was noted in each decade. One of the earliest procedures consisted essentially of cross hatching, crushing, and sawing the deviated cartilage and bone.[11] Reports at the turn of the Twentieth century dealt largely with septal operations performed through an oral approach.[12] and rebuttals by proponents of the nasal approach.[14,15] Then, because of the writings of such pioneers in septal surgery as Halle,[14] Killian,[15] Freer,[5,16,17] and Ballenger,[18] the intranasal technique of performing a submucous resection was placed on a firm footing. These authors also described their individual procedures for correcting septal spurs and deformities.

The dangers of saddling of the dorsum and retraction of the columella as a sequel to partial or total submucous resection created a period of discussion.[19,20] The problem of whether to perform septal surgery on children appeared intermittently in the literature.[12,14,21] Operating techniques for correcting caudal deflections of the nasal septum received much attention.[22,23,24,25,26,27,28,29,30,31,32] More recently papers have dealt largely with operative indications and the relationship of septal deformities to the broader field of rhinoplasty.[33,34]

Many articles pertaining to the etiology of septal deviations span the breadth of the literature.[35,36,37,38,39,40,41,42]

Developmental abnormalities, trauma in childhood or later life and delayed eruption of deciduous teeth have all been stated to be factors as the ultimate cause of deviations of the nasal septum.

In 1931 Neumann[43] discussed the mucoperichondrial and mucoperiosteal coverings of the nasal septum. He noted that, prior to that time, only Ballenger's textbook[44] contained diagrams of this anatomy. Lothrop[6] and Freer[1b] (see Fig. 3), and Aymard[45] also described these soft tissue reflections over the underlying rigid structures.

As late as 1858, after more than 200 studies had been devoted to the premaxillary bone in man and mammals; its existence as a normal structure in the skull of man was denied.[46] In 1869, however, a detailed developmental account of the premaxilla in man was published.[47] From this time on, there was no longer any doubt concerning the existence of the premaxilla in man. The embryological development of the premaxillary bones in the human is described comprehensively in two papers.[9,48]

b. Development of the Premaxilla in the Embryo: In man, the premaxillae are not observed at birth, because a delicate plate of the maxillary bones extends over them. It is for this reason that the very existence of a premaxilla (intermaxillary bone) in the human skeleton was a most controversial subject. The premaxilla is ossified from two centers placed side by side. The main center arises at the middle of the seventh week and the infravomerine center at the end of the tenth week.[48]

One of the more recent and excellent descriptions of the embryology of the premaxillae is that written by Ashley-Montagu.[9] The premaxillary structures contribute to the nasal septum, as the following quotation from this paper indicates:

"With the commencement of the ossificatory process, a number of premaxillary structures make their appearance and in the following order: *1.* A keel for the reception of the anterior-inferior portion of the vomer; *2.* the anterior floor of the nasal fossa; *3.* the anterior nasal spine; *4.* the ascending processes which form the walls and lateral borders of the pyriform aperture endofacially; and *5.* the palatine processes forming the anterior part of the palate, the median septum and the anterior and lateral walls of the incisive or nasopalatine foramen" (see Fig. 4).

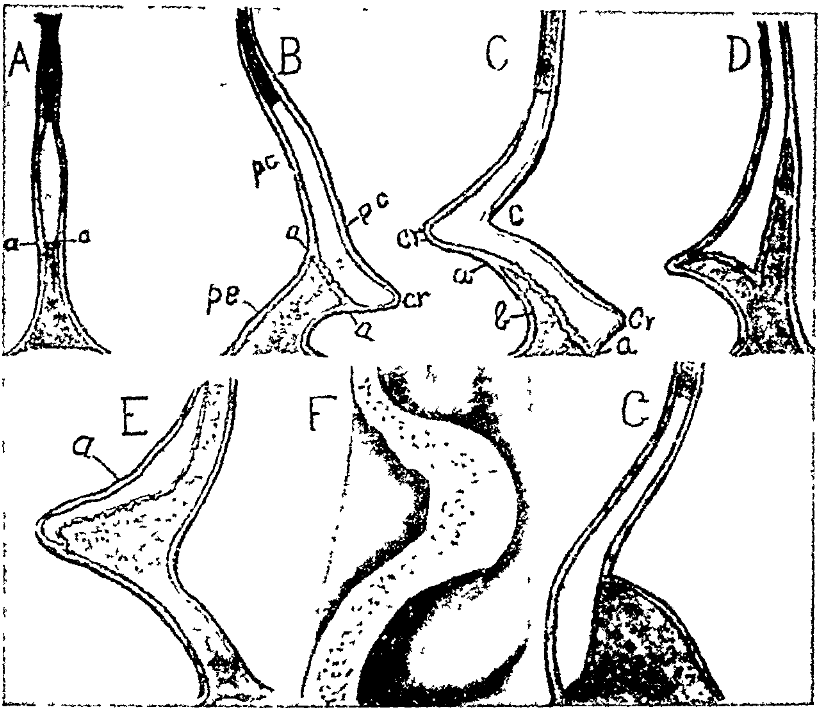


Fig. 3 A—Normal septum in vertical section showing perpendicular plate, cartilage and vomer, a a—vomero cartilaginous articulation showing crossing of blended periosteum and perichondrium from naris to naris.

B—Crest deflection in vertical section. a a—displaced vomero cartilaginous articulation slanting into naris of convexity; cr—cartilaginous crest; pc—perichondrium, pe—periosteum blending and crossing from naris to naris in the plane of the articulation a a.

C—Type of crest deflection with slanting of the vomero cartilaginous articulation into the naris of the concavity, with the formation of two crests and angular bending of the cartilage on itself to form a knee, c—concavity of the deflection; cr cr—the two cartilaginous crests; a a—line of vomero cartilaginous articulation with crossing of blended periosteum and perichondrium, b—vomer.

D—Unusual case where flanges of vomer were wedged open by the downward pressure of the growing cartilage to form a crotch

E—The junction of the vomer and perpendicular plate to form the bony V, a—processus sphenoidalis septi cartilaginei (caudal prolongation of Mosher), escaped from laterally opened joint and applied to side of vomer and perpendicular plate

F—A very massive bony V.

G—Type of crest deflection without overhang of vomer, producing a nearly straight septum in the narrowed naris with typical concavity in the other. Dotted line shows where the thick, wedge shaped vomer would ordinarily overarch the nasal floor. Such septa seem not to be in the median line, but really are so.

Mucoperichondrial and Mucoperiosteal Reflections of the Nasal Septum. (from **Freer**).

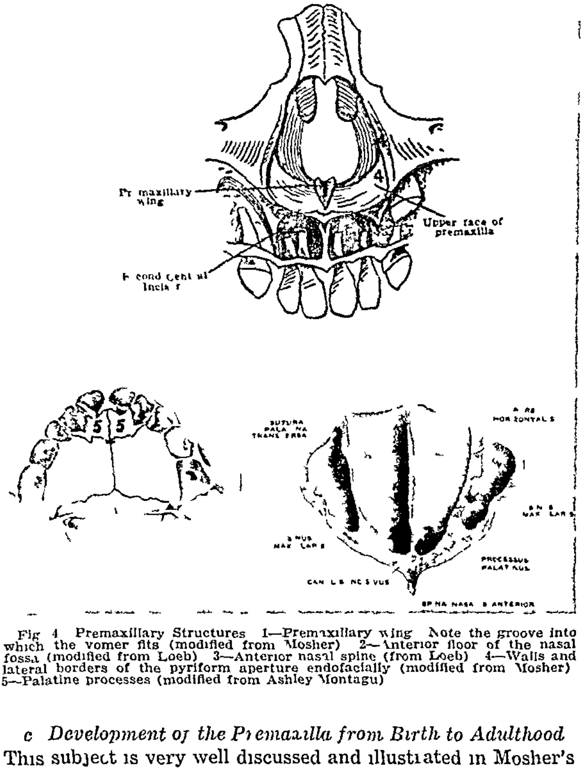
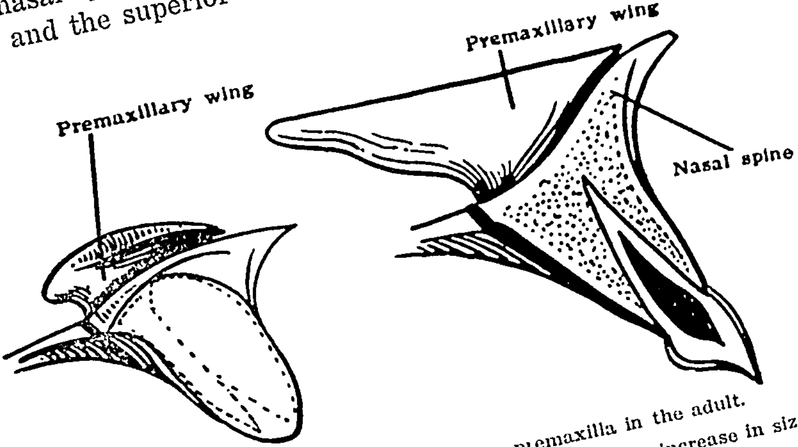


Fig 4 Premaxillary Structures 1—Premaxillary wing Note the groove into which the vomer fits (modified from Mosher) 2—Anterior floor of the nasal fossa (modified from Loeb) 3—Anterior nasal spine (from Loeb) 4—Walls and lateral borders of the pyriform aperture endofacially (modified from Mosher) 5—Palatine processes (modified from Ashley Montagu)

c *Development of the Premaxilla from Birth to Adulthood* This subject is very well discussed and illustrated in Mosher's publications[f, 36, 4] and is pertinent to the anatomical description of the premaxillary area

At birth there are two structures of the premaxillary bones that become component parts of the nasal septum, namely, the anterior nasal spine and the premaxillary wings

The nasal spine at birth is a pointed process that projects horizontally forward from the anterior edge of the upper face of the premaxilla (floor or the pyriform aperture). The superior surface of this is slightly concave. As the antra enlarge and descend with the eruption of the second tooth, the anterior nasal spine grows upward with the crests of the palate bone and the superior maxilla.



Premaxilla at birth. The premaxilla in the adult.

Fig. 5. The Premaxilla at birth and in the adult; note the increase in size and forward projection of the anterior nasal spine (from Mosher).

At birth the premaxillary wing projects obliquely upward and outward in the midline of the upper face of the premaxilla and is posteriorly contiguous to the anterior nasal spine. The premaxillary wings increase but little in size from birth to six years of age. From this time on they enlarge rapidly and are said to fuse with the tip of the vomer at 15 years of age (see Fig. 5).

The superior face of the premaxilla is flat like a plateau at birth and the premaxillary wing is firmly united to it by a slender neck and apparently is a part of it. In the process of maturing, the flat plateau disappears, and the superior face of the premaxilla slopes downward and backward at an angle of 45°, converting it into the shape of a moat.

In the region of the premaxillary wings the quadrilateral cartilage is thin and has its chief center of growth. The infant septum is cartilaginous except for the vomer and the premaxilla with its processes. The ossification time of the various parts of the nasal septum is well described by Spielberg.[42]

When fully developed, the vomer consists of two leaves of thin bone which are united below but are open and flaring above to form a V, or gutter. The premaxillary wings arise from the posterior half of the upper face of the premaxilla and duplicate the gutter formation of the vomer. The tip of the vomer rests in the wings. The nasal spines arise from the anterior part of the upper face of the premaxilla and likewise make a slight channel into which fits the tip of the premaxillary wings. To quote Mosher's[4] summary of the gutter formation:

> "The tip of the vomer rests in the gutter of the premaxillary wings, and the tip of the premaxillary wings rests in the gutter of the nasal spines, like the arrangement of the sections of the old-fashioned V shaped wooden drain" (see Fig. 6).

d. Mucoperiosteal and Mucoperichondrial Coverings of the Nasal Septum: In an original article concerning the mucous membrane reflections of the nasal septum, Neumann[43] published the first illustration of a histological section of this structure. He noted that most perforations following septal surgery occurred along the upper border of the vomer and occurred more frequently in the anterior portion than in the posterior. Another site where perforations had a tendency to take place was in the region of the crest and spine. If this complication of the submucous resection occurred more frequently in these areas, he reasoned, it must be due to the anatomical structures, and, therefore, he investigated the mucous membrane reflections of the nasal septum.

The membranous reflections surround three important sutures in the nasal septum: *1.* the suture between the quadrilateral cartilage and perpendicular plate of the ethmoid; *2.* the suture between the quadrilateral cartilage and vomer; *3.* the suture between the vomer and perpendicular plate of the ethmoid.

The mucous membrane is easily elevated over the suture line between the quadrilateral cartilage and perpendicular plate of the ethmoid, because the mucoperichondrium and mucoperiosteum are almost continuous except for a few loose fibers at the suture line.

The second suture line between the quadrilateral cartilage and the vomer presents a more intricate anatomical situation.

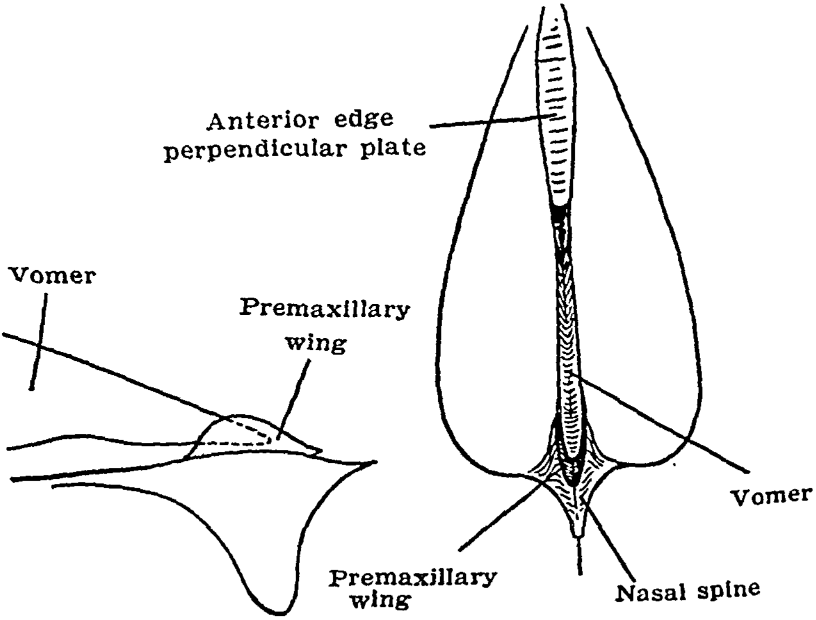


Fig. 6. The gutter formation; note the grooves and the arrangement of the anterior tips of the vomer, wing, and spine (from Mosher).

Here, the perichondrium overlaying the quadrilateral cartilage divides into an inner and outer layer. The outer layer is continuous with the outer layer of the mucoperiosteum over the vomer, but the inner one merges with the inner layer of the vomer periosteum and extends into and through the suture. This suture is wide and the inner layers which pass into the suture are thick.[43]

The suture line between the vomer and perpendicular plate of the ethmoid is quite narrow. Although the periosteum covering the perpendicular plate also divides into inner and outer layers, fewer fibers of the inner layer pass into the narrow suture line. This suture line is obliterated around 15 years of age, *thereby thinning out the inner layer of fibers.*[43]

Although Neumann did not make a histological study of crest formations, he thought the results would be the same as the normal septum. Killian[15] states:

> "The front end of the vomer and the crest are enveloped in their *own periosteum,* which, *therefore, covers their upper* borders after the cartilage has been resected from it."

The rigid structures underneath their soft tissue coverings are visualized with difficulty, because no boundary is visible where the soft tissues are reflected onto the bone. The edge of the bone cannot be seen but must be felt for with an instrument.[5]

MATERIALS AND METHODS.

Detailed anatomic studies were made on adult cadaver heads. Many patients, ages 20 to 40, were examined during septal operations performed by the author and other surgeons.

Six cadaver heads were allowed to soften in glycerin over a period of about one month. Two of the heads were sectioned sagittally just lateral to the midline. The remaining four heads were kept intact and dissected from the frontal aspect.

In one of the dissections each important anatomical part was covered with a different color in an effort to provide more contrast when photographing the structures and their anatomical relationships.*

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* A Primar Reflex camera with a F-3 5 Tessar lens was used in taking the photographs Lighting was obtained by means of a strobe light unit that fired through a close-up ring attachment. Metal extension tubes varying from 10 mm to 60 mm in length were used for close-up views. The most pertinent details recorded on the 2¼ x 2¼ inch size positive transparency could be cropped to the 35 mm size It was possible therefore, to obtain a telephoto effect without losing depth of field necessitated by the F15 6-F-8 exposure aperture of a 105 mm lens A polaroid filter was found to be necessary for reducing glare from the metal instruments and moist surfaces

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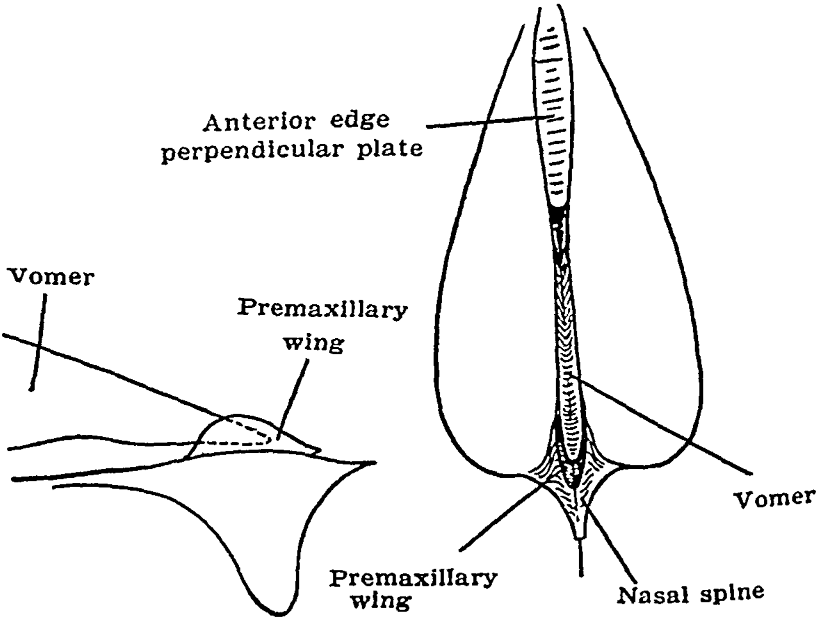

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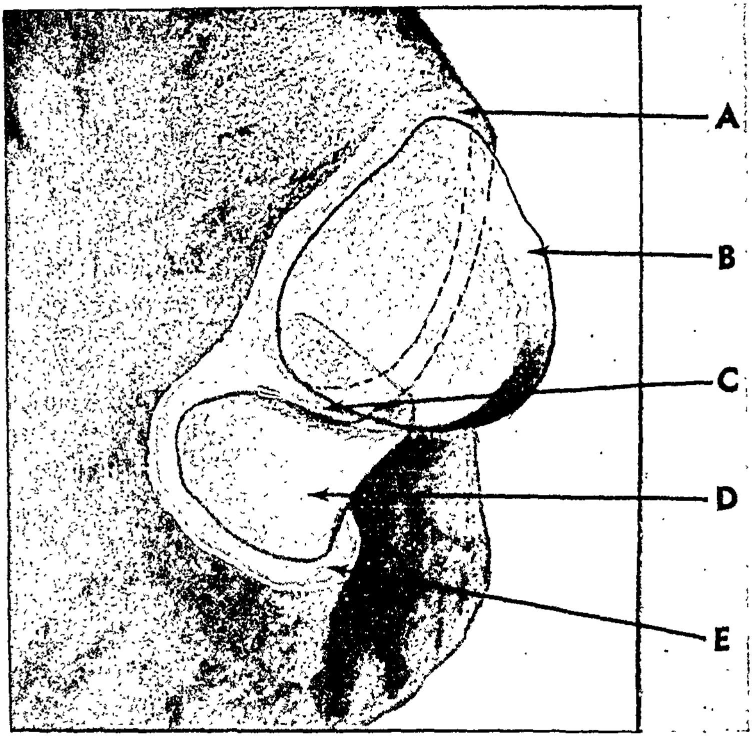

Fig. 7a. Perichondrial and Periosteal Envelopes of the Quadrilateral Cartilage and Anterior Nasal Spine (oblique view); note that the perichondrial and periosteal envelopes completely enclose their respective structures and fuse between their contact area.

A—Perichondrium; B—Quadrilateral cartilage; C—Fusion of perichondrium and periosteum; D—Anterior nasal spine; E—Periosteum.

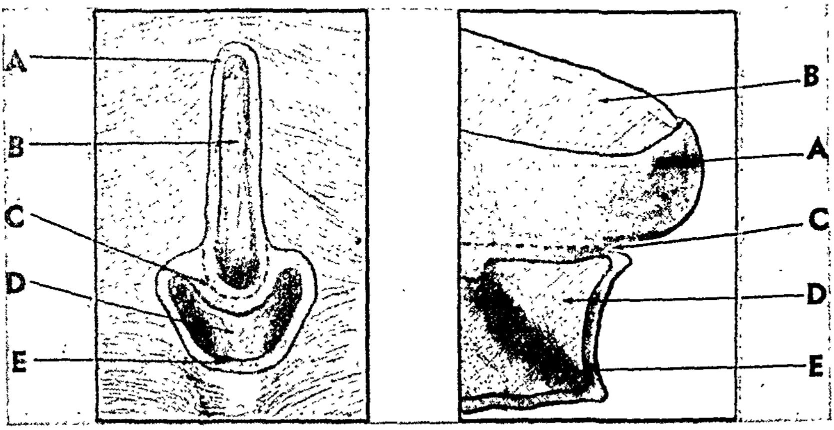

Fig. 7b and 7c. Perichondrial and Periosteal Envelopes of the Quadrilateral Cartilage and Anterior Nasal Spine (left drawing is a coronal view, and the right drawing is a sagittal view); note a type of joint capsule in the coronal view.

A—Perichondrium; B—Quadrilateral cartilage; C—Fusion of perichondrium and periosteum; D—Anterior nasal spine; E—Periosteum.

ANATOMICAL OBSERVATIONS ON CADAVER DISSECTIONS.

a. Description of the Adult Premaxilla: It will be noted from previous illustrations that in the adult premaxilla the anterior nasal spine is shaped like the prow of a ship. The anterior free end of the quadrilateral cartilage projects forward beyond the point of the prow. The caudal surface of this portion of the cartilage rests upon the superior face of the anterior nasal spine. In the region of contact between the quadrilateral cartilage and the upper face of the spine, the mucoperichondrium of the quadrilateral cartilage extends laterally and inferiorly to join with the periosteum of the wider anterior nasal spine. Perichondrium passes around the caudal surface of the cartilage and periosteum passes over the superior face of the spine. A type of joint capsule is formed within which side to side movement of the cartilage is possible (see Fig. 7a, b and c).

With hypertrophy of a wing, the cartilage is dislodged from its groove downward and laterally toward the floor of the nose. In this movement the cartilage carries with it its own perichondrium so that the caudal border is still encased within a perichondrial envelope. The perichondrial envelope is easily separated from the cartilage and the caudal portion of the quadrilateral cartilage may be levered upward.

In order to demonstrate the soft tissue reflections about the underlying rigid structures, the anterior prolongation of the quadrilateral cartilage is sectioned coronally at the point of the anterior nasal spine. The soft tissue coverings of the quadrilateral cartilage, premaxillary wings, and anterior nasal spine fuse with each other in this area.

The perichondrium on the medial side of the cartilage fuses with the mucoperiosteal envelope ascending upward and across the superior surface of the premaxillary wings (see Fig. 8).

The superior surface of the premaxillary wings is enclosed cartilage to the posterior portion of the cartilage. The attachment of the mucous membrane with the perichondrium is loose, and it is easy to separate the tissues in this plane. Separating the perichondrium in this plane may result in perforating the mucous membrane (see Fig. 9).

A study was made of the relationship of the free border of the cartilaginous septum and the premaxillary area. Arbitrarily, straight needles were inserted through the membranous septum at two points. The superior point was located at the anterior free end of the quadrilateral cartilage on a horizontal level with the attachment of the upper lateral cartilage to the quadrilateral cartilage (see Fig. 10). The inferior point was chosen at the free end of the quadrilateral cartilage on a horizontal level with the superior face of the floor of the pyriform aperture (see Fig. 10).

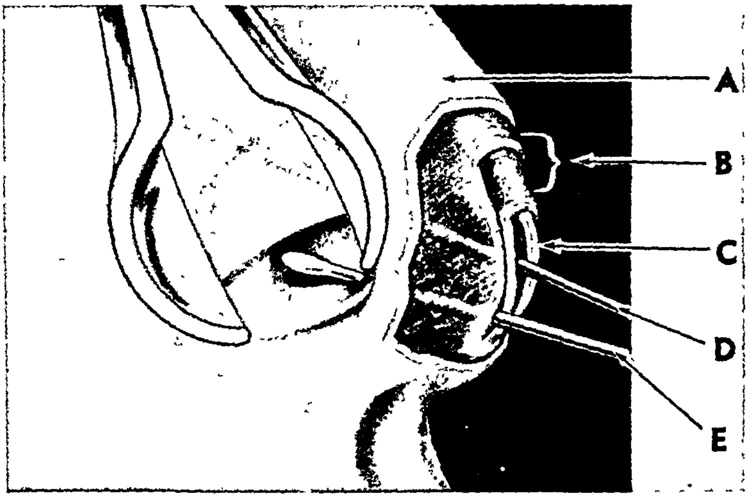

Fig. 9. Elevation of Mucoperichondrium; note the perforation when the extraperichondrial layer "E" of the mucoperichondrium is elevated.

A—Skin; B—Mucoperichondrium; C—Anterior free end of quadrilateral cartilage; D—Elevator under mucoperichondrium; E—Elevator in extraperichondrial layer.

On dissection, the needles crossing the anterior free end of the quadrilateral cartilage were designated with two colored marks (see x and y in Fig. 10a). It can readily be seen that

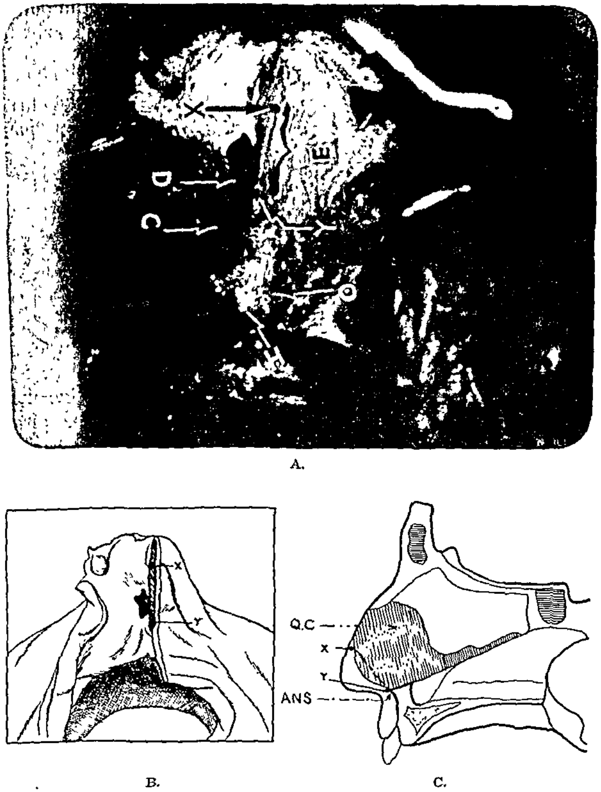

Fig. 10a, b and c. Limitations of intranasal septal incision. An imaginary line connecting x and y in frontal view Fig. b; corresponds with bracketed area E in frontal view Fig. a; this also corresponds to the line x-y in lateral view Fig. c; note that the premaxillary area is caudal to points y as well as bracketed area E.

Fig. A. H. Anterior nasal spine; O. Periosteum of anterior nasal spine; C. Floor of nasal vestibule; D. Perichondrium; E. Anterior free end of qudrilateral cartilage corresponding to line x-y.

Fig. B. is a schematic line drawing of Fig. a.

Fig. *C.* Q.C.—Quadrilateral cartilage; ANS—Anterior nasal spine.

the antero-caudal part of the quadrilateral cartilage, the point and the median ridge are not visualized caudal to a line bracketing the two points x and y.

If x and y are connected as with any intranasal incision, it is apparent that the premaxillary area with its entire periosteal envelope is not exposed (see Figs. 10b and 10c).

SURGICAL ANATOMY.

This division of the subject is concerned with an application of the descriptive anatomy of the antero-caudal end of the nasal septum to surgery on this area. One of the frequently used surgical techniques that utilizes to advantage a knowledge of this area is the Cottle incision in the anterior free end of the nasal septum, and his procedure for undermining the base of the nose. The details for making the incision are well discussed and fully illustrated in Cottle's publications.[2,27,31]

It is important to re-emphasize the fact that the elevation of the mucoperichondrium from the quadrilateral cartilage should begin precisely in the plane where these two structures are contiguous. The bluish color of the bare cartilage should be carefully looked for before elevation of the mucoperichondrium progresses in a posterior direction. If the dissection is carried backward in a split layer of the mucoperichondrium, a perforation in the mucous membrane will result (see Fig. 9).

After the incision is made, a varying portion of the septal operation may be performed. Then, a curved, Knapp scissors is inserted into the lower edge of the incision. The back of the scissors is held firmly against the median ridge of the anterior nasal spine as the blades are opened up and soft tissues are spread in order to create a pocket. A speculum is next inserted into the pocket and placed into a position to straddle the median ridge. A longitudinal incision is next made into the soft tissue on each side of the median ridge and the periosteum elevated from the anterior nasal spine. This procedure should be limited from the point and median ridge of the nasal spine anteriorly to its base posteriorly, since extensive upraising of the periosteum would lead laterally along the floor of the pyriform aperture. With the anterior nasal spine cleaned of its soft tissue covering, the perichondrial envelope of the antero-caudal end of the quadrilateral cartilage [illegible] exposed for further dissection. The perichondrium can be elevated easily from this part of the quadrilateral cartilage [illegible] the cartilage resected.

The premaxillary wings covered with *mucoperiosteum* then begin to come under direct vision. As more of the ventral edge of the quadrilateral cartilage is removed in an anterior-posterior direction, the premaxillary wings are also more exposed. Frequently the caudal-ventral edge of the quadrilateral cartilage may be removed by insinuating a perichondrial elevator underneath this surface of the cartilage. By exerting a [illegible] upward motion, the cartilage can be lifted out of its perichondrial envelope, and, thus being cleaned of its perichondrium, it may be eliminated with a biting forceps.

One can incise the mucoperiosteal sheath covering the premaxillary wing through its anterior edge and superior surface and then continue the elevation in an antero-posterior direction. This can be done, even though the periosteum is somewhat adherent to the underlying bone. After freeing the underlying rigid structures from their soft tissue coverings, the bony portion may be handled by several different methods. It may be infractured, split sagittally with a chisel, fractured and removed with a Koeffler punch, or detached from the maxilla with a 4 mm. chisel and segments removed with a forceps.

Another method of uncovering the rigid structures from their soft tissue reflections is as follows: A Pierce elevator, or similar instrument, is inserted on the concave surface between the mucous membrane flap and the perpendicular plate of the ethmoid. The instrument then is carried downward to pierce the vomerine suture fibers from above. The inner fibers are loose at the suture of the perpendicular plate of the ethmoid and the vomer, and are easily perforated. The mucous membrane is raised down to the crest of the superior maxilla. The flat surface of the Pierce elevator is held firmly against the vomerine bone and is carried forward toward the caudal end of the nasal septum. As the instrument sweeps forward, the mucoperiosteum covering the vomer is separated easily from that underlying bone.

The elevation is more difficult at the wing, and gradually more force must be exerted in pulling the instrument forward. The fibers will suddenly give way and the elevator appears in the gutter of the anterior nasal spine. The premaxillary wings are chiseled free and splintered upward. The bony pieces are then removed from the mucoperiosteum covering their convex side by grasping a free edge of the bone with a Ferris-Smith forceps and gradually rocking them loose. The bone is taken from its covering rather than elevating the covering on the convex side.[1]

The floor of the pyrioform aperture is quite wide and this fact is of some importance when chiseling free the premaxillary wings. When a 4 mm. or 7 mm. sized chisel is used as the instrument of choice, caution must be exercised to avoid driving the blade into the upper face of the floor. Properly placed, the blade should clear the inner nasal edge of the pyriform floor as it is driven into the premaxillary wing; also, to prevent hemorrhage, a determined effort should be made to avoid striking the vessels coming up to the floor of the nose through the nasopalatine foramen.

DISCUSSION.

Considerable attention is given to the "gutter formation" of the premaxillary wings, vomer, and nasal spine. The various soft tissue envelopes that cover the rigid underlying structures result in three conjoined compartments: the anterior nasal spine, the premaxillary wing, and the antero-caudal end of the quadrilateral cartilage.

Certain features of the mucoperichondrial and mucoperiosteal coverings of the nasal septum indicate the possibility of connecting the conjoined anterior compartments with the posterior portion of the elevated mucous membrane flap. The main feature of the coverings seems to be the ability to penetrate the inner mucoperiosteal fibers at the vomer and per-

perpendicular plate suture line from above downward toward the floor of the nose and then by coming forward on the concave side to elevate the coverings from the vomer to and into the maxillary area.

In performing a submucous resection, it was generally ed, at the beginning of the Twentieth century,* that better perspective was obtained with the oral approach than with the nasal method. The pioneers of the submucous resection rejected the oral approach for such reasons as poorer healing, prolonged post-operative discomfort, and longer operating time, and adopted the intranasal method.

The surgical approach to the anatomical structures of the antero-caudal portion of the nasal septum should result in a visualization of these structures that more closely resembles that obtained with the discarded oral technique.

It is obvious from a study of a frontal dissection on a cadaver head that any of the standard submucous resection incisions fail to give easy access to the entire periosteal envelopes of the premaxillary area. The two needle perforation marks, when projected from the frontal to an intranasal position and connected intranasally by an imaginary line, denoted the limitation of any standard submucous resection incision.

It would seem desirable to select a surgical technique that will allow the surgeon to expose adequately the premaxillary area through an intranasal incision. The Cottle septal incision and technique of undermining the base of the nose logically will not injure the respiratory epithelium, and will allow better exposure with simpler access to the bony portion of caudal basal spurs and deviations of the nasal septum. There is more postoperative discomfort because of the greater exposure, but the ease of surgical performance and lessened chance of encrusting perforations far outweigh this disadvantage.

SUMMARY AND CONCLUSIONS.

1. A detailed anatomical study on cadavers and patients of the mucoperichondrial and mucoperiosteal reflections of the antero-caudal end of the quadrilateral cartilage, premaxillary wings, and anterior nasal spine is presented. The anatomical information concerning the conjoined compartments of these structures would imply an individual surgical approach to each compartment; partial or total removal, and/or realignment of the rigid structures. With such exposure, continuation of the elevated posterior mucous membrane covering with the anterior conjoined compartment can be accomplished.

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* See Review of Literature, page 1000.

2. It was confirmed that the surgical technique of undermining the base of the nose gives an excellent exposure to the premaxillary area.

3. The anatomical findings agree with observations previously reported.

ACKNOWLEDGEMENT — We gratefully acknowledge the editorial advice of Dr. Ben H. Senturia and Dr. Joseph Ogura, Department of Otolaryngology, Washington University School of Medicine, St. Louis, Missouri; the technical assistance of Mary Holliway and Felecia Spurrier; and the colored drawings of Don Langeneckert.

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SURGERY OF THE LARGE ARTERIES OF THE NECK.*†

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In the various fields of surgery, many noteworthy advances have been made during the past few decades. If we exclude the progress made in the control of bacterial infection by antibiotic substances, then no greater contribution to the practice of the surgery of trauma has been made than the development of the technical and supportive methods which make possible the successful replacement of large arteries in man.

Most of the serious consequences of wounds of violence depend to a large degree upon the kind and extent of the damage to the underlying large blood vessels. Methods for controlling the excessive loss of blood from operative or accidental wounds of large blood vessels, particularly in the neck, have interested surgeons almost from the dawn of medical history. A great variety of procedures has been suggested as a means of reducing the loss of blood from such wounds and, at the same time, maintain an adequate supply of arterial blood to the brain. The maintenance of an adequate flow of blood to the brain in such patients after trauma to the great arteries of the neck, therefore, becomes the responsibility of the surgeon. If the injured artery cannot be reconstituted by careful repair of the arterial wall, then the surgeon must be able to restore the continuity of the artery by replacing the damaged segment with a suitable graft of autogenous, homologous, or synthetic blood vessel of the proper caliber.

The problems concerning the surgery of the large arteries of the neck can be divided roughly into two main categories: first, those problems associated with injury to arteries or veins in wounds of violence, or unavoidable injuries incident to the surgical removal of large tumors of the neck; second, those problems associated with restoration of the arterial circulation to the brain in patients with congenital anomalies of the blood vessels of the neck, or some acquired disease of these large blood vessels.

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* Read by invitation at the meeting of the Middle Section of the American Laryngological Rhinological and Otological Society, [illegible] Ohio, January 16, 1956

† From the Department of Surgery College of Medicine, University of Cincinnati and the Lucie Rawson Laboratory for Vascular Research, Cincinnati General Hospital

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Non-penetrating injuries to the neck may cause rupture of medium sized arteries or veins and give rise to large hematomas, with either extensive extravasation of the blood into the subcutaneous tissues of the neck, head and chest, or a pulsating hematoma which compresses vital structures in the neck. Early decompression of the fascial compartments of the neck, with or without tracheostomy, may become imperative if the accumulation of the blood in the neck continues to take place.

Penetrating injuries in the vicinity of the large arteries of the neck must always be considered as grave, even though no external bleeding is taking place at the time the patient reaches the hospital. A small soft clot may stop bleeding for a few hours after a tangential injury to the carotid arteries, but secondary hemorrhage is certain to follow sometime later. Tangential injuries to large arteries may result in exsanguinating hemorrhage without premonitory signs or symptoms, or extensive thrombosis of the important artery may take place. Tangential injuries of large arteries in the fresh state can usually be repaired without difficulty by sutures. Injuries of this type, if left untreated and if the patient recovers spontaneously, will always give rise to a false aneurysm or a pulsating hematoma. These complications are much more difficult to cure by surgical operation than is the original tangential injury to the artery. We are of the firm opinion that the surgical exploration of all penetrating wounds of the neck is the procedure of choice.

The segmental arteriospasm which frequently complicates injuries to large arteries of the neck can only be treated adequately by bathing the injured artery in 2 per cent papaverine solution after it has been exposed at operation. Novocainization of the regional sympathetic ganglia is of no value in overcoming the cerebral ischemia or ischemia of the muscles of an extremity from this type of segmental arteriospasm. Hemiplegia usually results from the cerebral ischemia if this type of arteriospasm is not promptly relieved, and autolysis of skeletal muscle with the production of Volkmann's contracture may result in extremities.

With modern supportive methods, it is no longer necessary, and rarely desirable, to replenish the lost blood to its full extent and thus raise the systemic blood pressure to its normal level. This only encourages further hemorrhage by dislodging a soft clot at the site of injury to the large artery. After the defect in the artery has been repaired, then the re-establishment of normal blood volume is indicated.

Much of the surgery upon large arteries should be classified as elective surgery, and careful study and preparation of the patient is essential if the surgeon hopes to obtain lasting relief without serious complications from his operations. Successful arterial surgery depends upon adequate exposure of the injured arteries by properly placed incisions of reasonable length and careful dissection of the tissues with as little bleeding as possible.

PRINCIPLES GOVERNING SURGERY OF LARGE ARTERIES.

Operative procedures which are designed to re-establish the continuity of large arteries must be performed soon after the initial injury if restoration of the arterial circulation is to be accomplished.

The successful repair of a wound of any large artery is a difficult task, and the surgeon must constantly bear in mind that blood vessels carry a remarkable fluid which normally has a tendency to congeal. The formation of clots of blood within large arteries after they have been repaired results in the failure to re-establish the functional activity of the artery. Most arteries which require repair are either diseased or damaged by trauma; consequently, the problems are different from those which concern the surgeon who plans to suture a normal artery of man or some experimental animal. In addition to the problem of intravascular thrombosis in the injured or repaired artery, the surgeon must concern himself with the adequacy of the arterial circulation beyond the point of injury to the artery. If our surgery is to be successful, we must understand thoroughly the mechanisms which control the peripheral arterial circulation.

Collateral Arterial Circulation. The advantages of knowing the behavior of the peripheral circulation in the extremity or in the brain after the main nutrient artery has been severely injured should be obvious to all of us. It is our duty to ascertain by actual tests what alterations in the efficiency of the peripheral circulation will take place if the major arterial trunk to that part is permanently and suddenly occluded during any surgical operation. When the collateral arterial circulation is active enough to keep the distal parts of the extremity in proper circulatory balance in spite of injury to or occlusion of the main artery, we believe it is inadvisable to spend the time and labor necessary to perform a successful repair of such an artery, since a single ligature on the proximal and distal ends of a completely severed artery will usually suffice to prevent further extravasation of blood into the muscles or to the outside. If temporary occlusion of the injured artery causes the extremity to remain cold and cadaveric in color, in spite of a satisfactory environmental temperature, it would be wise to postpone any surgical operation upon that artery. If, however, postponement of the operation is not possible because of the nature of the arterial injury, the surgeon must resort to every available means of restoring the continuity of the injured artery and of enhancing the collateral arterial circulation.

It is imperative to determine the patency of the communicating pathways between the vertebral arteries and the carotid arterial circulation before permanent obliteration of the internal or common carotid artery is undertaken. We advocate the use of local anesthesia when arteries of the neck are to be exposed by operation, in order that the cerebral effects following temporary occlusion of the carotid arteries may be studied.

If the patient shows no ill effects after 15 to 30 minutes of temporary occlusion of the internal carotid artery, we can be certain that the collateral arterial circulation of the brain is adequate and permanent occlusion of that artery will be tolerated without immediate complications.

The fatal cerebral ischemia which occasionally follows the ligation of the common carotid artery is the result of preexisting anatomic anomalies of the circle of Willis or ascending arterial thrombosis. We have followed the suggestion of Matas and have always applied an aluminum band when the common carotid artery is to be occluded, while the external carotid artery is always obliterated by a ligature. If the patient should show signs of cerebral ischemia within several hours after the operation, the aluminum band can be removed from the common carotid artery with complete restoration of the circulation.

Secondary Vasospasm. The sudden interruption of the flow of blood through a large artery usually is attended by marked vasospasm in the entire peripheral vascular bed of that artery; consequently, the degree of arterial insufficiency is often out of proportion to the local injury of the main artery. This secondary vasospasm must not be confused with segmental arteriospasm which is myogenic in origin. Reflex vasospasm can be relieved by the use of vasodilating drugs, such as the oral administration of alcohol, the intravenous administration of papaverine or etamon, or by the novocainization of the regional sympathetic ganglia. Occasionally after injury or repair of a large artery, the peripheral ischemia is so great that a sympathetic ganglionectomy or ramisection must be performed in order to bring about proper balance in the peripheral circulation.

Locally applied heat increases the local metabolism of the tissues but does not greatly enhance the flow of arterial blood to the distal parts; consequently, the deficiency of oxygen to the tissues becomes greater and local death of tissue frequently ensues.

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OPERATIVE PROCEDURES UPON LARGE ARTERIES.

Contrary to general belief, there are still many dangers associated with the aseptic ligation of large arteries. Asepsis has greatly lessened the dangers of secondary hemorrhage and the secondary re-establishment of the lumen after ligation of a large peripheral artery; nevertheless, these dangers still must be kept in mind when large arteries of the trunk or neck are ligated or when the surgeon must deal with abnormally large and thin-walled peripheral arteries. Permanent occlusion will be more satisfactory if a wide segment of the artery is included in the ligature or the band, and also if the ligature or band is applied in such a way as to cause the least mechanical damage or fracture to the arterial wall. The process of necrosis of the artery will then take place slowly, and complete substitution of dense fibrous tissue for the necrotic tissue ultimately will take place.

Permanent Occlusion of Large Arteries. Permanent occlusion of large arteries can be accomplished by ligatures, cotton tape, metallic bands, strips of heavy fascia, or plugs of heavy fascia anchored within the lumen of the artery. It is necessary to remember that the portion of artery included in the ligature must undergo necrosis and the occlusion is made permanent by the substitution of living fibrous tissue for the necrotic tissue. For this reason, we do not advise the use of absorbable materials for permanent occlusion of large arteries, because these materials may stretch or loosen and thus subject a necrotic portion of the artery to the pressure of the blood within the artery. It is important to use some unyielding material, such as silk, metal or cotton tape.

The rate of necrosis of the arterial segment beneath the ligature varies directly with the size of the ligature and the size of the artery. A small ligature about a large vessel runs infinitely more risk of cutting through, with the result of secondary hemorrhage or restoration of the lumen of the artery, than does a large ligature or piece of cotton tape. We believe the ligature should vary in size directly with the size of the artery. When we are dealing with very large arteries, we believe that a transfixing suture of medium silk should be used to prevent the tape from untying or being blown off the end of the artery.

It is well known that the cutting effect of ligatures on large arteries is greatly reduced if the artery is divided instead of ligated in continuity. We, therefore, advocate, whenever it is possible, the division of an artery which is to be permanently occluded by ligature. Occasions do arise when it is not advisable to divide the artery completely.

Aluminum bands also may be used for the permanent occlusion of arteries. These bands consist of strips of aluminum stock of 0.573 mm. thickness (23 Brown & Sharp's metal gauge) about 5-16 inch wide, for the occlusion of an artery the size of the carotid artery. Strips of autogenous fascia have been used to occlude arteries and pieces of cellophane have been applied about arteries to cause gradual occlusion by fibrosis. Both these methods have practical value.

The fundamental process of pressure necrosis by the ligature and the repair or replacement of this injured tissue is essentially the same in infected wounds as in clean wounds, except for the degree and rapidity with which these changes progress. The dangers of secondary hemorrhage are considerably greater after occlusion of a large artery in an infected wound. The infection hastens the dissolution of the segment of vessel beneath the ligature, frequently causes liquefaction of the blood clots, and greatly retards the process of repair. We believe that this is more reason to use unyielding ligature material for the obliteration of large arteries in infected wounds.

When a major artery and its concomitant vein must be ligated, it is important to ligate separately the main artery and the main vein. If the ligature includes both artery and vein, an abnormal communication between the artery and vein might result as the vessel walls beneath the ligature undergo the usual process of necrosis and replacement with fibrous tissue. The development of an abnormal arteriovenous communication in the neck might lead eventually to serious complications.

After extensive injury to both artery and vein by gunshot wounds or stab wounds, it is equally important that the proximal and distal ends of the injured artery and vein be isolated and ligated separately to prevent the subsequent development of an abnormal arteriovenous communication.

Suturing of Large Arteries. Since the successful repair of a wound of any large artery depends upon the prevention of secondary thrombosis at the site of suture, it is fortunate that we have available chemical substances which retard the intravascular clotting of blood. The most important of these substances is heparin. It is a natural anticoagulant which is normally present in the blood in small quantities. The prolongation of the clotting time of blood following the administration of heparin depends entirely upon the amount of the substance administered, and there is no phase when the clotting time cannot be altered by the injection of an active preparation of this substance. It is possible, therefore, to use heparin locally in the irrigating solutions at the site of the operation upon a large artery, and it also can be given parenterally to reduce the clotting power of the blood in the entire blood circulation. Heparin in doses of 50 mg. to 100 mg., given intravenously every four hours, usually prolongs the clotting time of the circulating blood to about 15 minutes. If the effect of heparin is to be neutralized at any time during or after a surgical operation, this can be done promptly by the intravenous injection of protamine sulphate.

If heparin is to be used following any surgical operation, careful attention should be paid to the hemostasis at the time of operation. It is wise to wait about four hours after the operation before therapeutic amounts of heparin are given. Careful observation of all patients receiving heparin therapy is essential and heparin should not be given to patients with known blood dyscrasias or obstructive jaundice. Heparin has proved to be a valuable adjuvant to the vascular surgeon. For prolonged reduction of the clotting power of the blood, we feel that dicumarol has many advantages over heparin, since it can be given orally once each day.

The most important cause of intravascular injury is the lodgment of a blood clot which has been carried distally from the heart, or from some larger peripheral artery, into an important secondary peripheral artery. The sudden damage to the intima of the peripheral artery provokes marked reflex vasoconstriction in the entire vascular bed. The stagnation of blood brought about by the vasoconstriction favors progressive thrombosis within the vascular system of the entire extremity. Damage to the intima takes place easily, and secondary thrombosis at this site begins within a few hours after the embolus has lodged. If there is evidence of progressive intra-arterial or intravenous thrombosis, it is imperative that we use anticoagulants to retard the clotting mechanism of the blood. The decision as to whether the acute arterial occlusion should be treated by conservative means (such as vasodilating drugs, optimum resting level, paralysis of sympathetic nerves, and passive vascular exercises), or whether surgical intervention should be resorted to without further delay, is still a matter which requires keen judgment based upon wide experience.

We believe acute occlusion of arteries of the neck should be considered as a surgical emergency, and every means of promoting an adequate exchange of blood through the collateral arterial pathways should be instituted without delay. General conservative measures, in addition to early heparinization of the patient, will result usually in satisfactory circulatory balance; nevertheless, the surgeon must be ready to perform an arteriotomy and remove the embolus from the lumen of the artery if an adequate arterial circulation to the brain cannot be brought about immediately. It also must be borne in mind that the operation of embolectomy is an extremely delicate one and, if great care is not exercised in the removal of the intravascular clot, the added damage to the intima will result in subsequent thrombosis and occlusion of the artery. The site of lodgment of the embolus must be determined accurately and the artery exposed with as little surgical trauma as possible. The operation is best done under local anesthesia with adequate exposure by properly placed skin incisions. As soon as the artery has been freed of the embolus, it should be thoroughly irrigated with a 1:500 solution of heparin in normal saline solution. The intima and the media of the artery should then be closed by a running suture of very fine silk. When further clotting of the blood within the artery takes place, especially in older people, the problem becomes more difficult. Heparin also should be administered under these circumstances, but it must be remembered that it will not affect thrombosis which has already taken place.

BRIDGING DEFECTS IN LARGE ARTERIES.

The practical aspect of bridging defects in large arteries, especially arteries where greater intraluminal pressure exists, is of great concern to us. To provide additional strength in such a vascular graft, surgeons naturally turned to the use of fresh or preserved segments of large arteries from other human beings, even though homologous grafts of all other living tissues have failed to retain their original function when transplanted into man. The technical, biologic, and legal problems incident to obtaining, sterilizing, storing, and implanting such homologous arterial grafts proved to be great obstacles to their universal use. Volumes of scientific reports have accumulated on this aspect of the subject alone.

It was only natural for all of us to turn again to the fundamental aspects of the problem and search for more suitable and more easily obtainable substances. Again the products of modern metallurgy, body tissues, animal secretions, and organic chemical compounds were fashioned into tubes and used to replace large arteries in animals, with varying degrees of success.

Soon thereafter the chemists began to make available, for experimental use, several varieties of plastic sheets and fibers which rival silk and metals in strength, smoothness and inertness. It was soon demonstrated that the body tissues tolerate these plastic substances even better than fibers of animal origin. Just as surgeons of a generation ago used silver tubes, magnesium tubes, and even glass tubes, so now we began to use tubes of impervious plastic materials, with or without a lining of autogenous tissue, to bridge defects in large arteries.

These, too, proved reasonably successful, and because of their smooth lining and their blood-repellent properties, they functioned as replacements for segments of large arteries of the neck or extremities, with relatively few complications. Hufnagel, Shumacker and their associates have been interested in the use of impervious plastic materials, and their studies have added much to our knowledge of the biologic reactions to these synthetic substances.

Our past experience in the field of vascular surgery convinced us that this problem is a physiologic, rather than an anatomic, one. Physiologically speaking, defects in large arteries must be bridged by autogenous tissues or by an elastic frame of some inert and pervious material through and about which the body cells can proliferate and form permanent protective layers.

Voorhees, Jaretzki and Blakemore offered a truly physiologic approach to this problem when they demonstrated that closely woven cloth of some of the synthetic fibers permitted the autogenesis of arterial coats of fibrous tissue, with the plastic cloth tube serving as the elastic layer of the new artery. This, we believe, is a fundamental contribution to the problem of arterial replacement, and our experience in the laboratory with fine meshes of various synthetic fibers has convinced us that "arteriogenesis" by this means is not only possible but may be a practical solution for some of our most perplexing vascular problems.

We believe that the remarkable ability of the plastic cloth to bring about "arteriogenesis" depends upon the physical property of being pervious as well as elastic. We have used cloth woven from various synthetic fibers; namely, Nylon, Dacron, Orlon, and Vinyon-N.

Sections of the aorta at the junction with these plastic tubes have shown that the Orlon or Dacron is very quickly covered by a firm layer of fibrous tissue which is an extension of the outer coats of the aorta. The inside lining of the plastic tube is made up of a thin layer of cells which appear to be fibroblasts. These fibroblasts are seen to extend from the outer layers through the meshes of the plastic cloth. Within six days a well-formed layer of these fibroblasts lines the plastic tubes, and this layer is continuous with the intimal lining of the aorta above and below the graft. The blood-repellent properties of these plastic fibers could be responsible for the fact that thrombosis does not take place immediately, as it does in tubes of metal and animal products.

There is very little cellular reaction around the fibers of Orlon or Dacron, while there are occasional giant cells about the silk sutures which are used to hold the graft in place. We are now interested in using Orlon or Dacron fibers to fix the graft in place.

COMMENT.

In our work during the past two years, we have used Orlon and Vinyon-N in the form of patches of cloth and tubes made of the cloth or woven tubes of greater thickness and strength. The bleeding through the meshes of the cloth of Vinyon-N, which has been mentioned by Voorhees, Shumacker and Hufnagel, is related to the kind of weave, as well as to the physical character of the fiber itself. Orlon fiber is smoother and finer and can be woven tighter; consequently, it does not permit "bleeding through the mesh" and has, therefore, been preferred by many of us. Both Orlon and Dacron cloth are well tolerated by the body tissues, and both possess a degree of elasticity which closely approximates that of the elastic layer of the aorta. The hand-fabricated tubes make functional segments, but several investigators (Voorhees, Blakemore, Hufnagel and ourselves) believe that seamless tubes of these fibers, in various sizes, shapes, and with desired branches for renal arteries, common iliac arteries, and even smaller arteries, and which are now available commercially, are ideally suited for replacement of segments of human arteries.

The histologic studies indicate that the pervious and elastic matrix has served as a framework upon which body cells, principally fibroblasts, from the periarterial or retroperitoneal or retropleural tissues, have been able to proliferate through the meshes. This gives a smooth layer of fibroblasts, which serves as the intima, with heavy coats of fibrous tissue outside the plastic mesh to serve as protective coats of the new artery. We believe this is arteriogenesis and should be an ideal method for replacing even long segments of the aorta, since the functional layers appear to be developed from without inwardly. The new intima is not produced by ingrowth of cells from the artery at either end of the graft. This method is worthy of more serious study and further application in man.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Arrangements have been completed for the joint meeting of the North Carolina Society of Eye, Ear, Nose, and Throat, and the South Carolina Society of Ophthalmology and Otolaryngology September 17, 18, 19, 1956. Headquarters will be the George Vanderbilt Hotel, Asheville, North Carolina.

An unusually attractive program has been arranged, and a large attendance is anticipated.

Asheville, North Carolina, is in the mountains of Western North Carolina, and is a particularly beautiful spot in this season of the year.

For further information write Roderick Macdonald, M.D., Sec. and Treas., 330 East Main Street, Rock Hill, S. C.

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Better understanding of the indications for the operation and improvements in technique by Henry Butlin and Sir Felix Semon in England resulted in its reinstatement after its first loss of favor. It was practiced successfully by Sir St. Clair Thomson in England and by Chevalier Jackson and others in the United States during the early years of this century. In 1922 Chevalier Jackson,[4] recognizing the fact that lesions reaching the anterior commissure should be treated differently from those restricted to the middle third of the cord, presented his well known "anterior commissure technique," according to which the thyroid cartilage, instead of being divided in the midline with shears ("clipping technique"), was divided with a handsaw or a motor driven circular saw, in order not to cut through or crush the tumor. In a paper on laryngofissure presented before this Society by one of us[3] in 1940, as a candidate's thesis, a series of 50 operations was reported: 32 of them done by the clipping technique, and 18 by the Jackson anterior commissure technique. This latter technique was modified by Clerf[5] and subsequently by Kemler[6] and by Broyles.[7] Broyles added valuable scientific support for the concept of cartilage removal at the anterior attachment of the cords by his anatomical studies of the anterior commissure tendon.

At the Chevalier Jackson Clinic of Temple University Hospital we still do the classic laryngofissure operation in cases of cancer involving the middle third of one vocal cord, but we use a modified form of the anterior commissure operation in cases in which the lesion reaches the anterior commissure or crosses over to the opposite cord. In such cases we do a *partial laryngectomy* with removal of varying amounts of underlying cartilage, an operation perhaps similar to what has been called by Leroux-Robert[8] "anterior fronto-lateral laryngectomy." Even if the lesion does not quite reach the anterior commissure, we generally remove the anterior three-quarters

EVOLUTION OF SURGICAL TECHNIQUE IN THE TREATMENT OF CARCINOMA OF THE LARYNX.*†

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and
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Though this title sounds historical, we are not going to undertake a detailed review of the history of the surgical treatment of carcinoma of the larynx. A full account of the developments of the earlier years may be found in the literature.[1] The grim story of the early days of both laryngofissure and laryngectomy is interesting to read and provides the proper background for the best understanding of the more recent trends.

Endoscopic excision is the most limited form of partial laryngectomy. Its technique has not undergone any remarkable changes and probably the most important thing to say about it is to reemphasize the importance of the most careful selection of cases, according to the advice of LeJeune.[2]

"Laryngofissure"[3] (thyrotomy) is a term which should probably be restricted to the operation of simply splitting the larynx (or rather the thyroid cartilage) in order to gain access to the interior of the larynx for the purpose of removing the tumor and surrounding normal soft tissue. This operation is suitable for very early tumors of one vocal cord, but it is the general recognition of its limitations, and the consequent indications for its selection as a treatment method that has so securely established its place in the treatment of cancer of the larynx. The disrepute into which this operation fell during

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proved technique, the operative mortality rate has fallen to almost zero. In our clinic the only operative death occurring in the past 15 years, in 255 laryngectomies, was due to cardiac arrest, in a patient with complete heart block. While, of course, the operation must be admitted to be a disabling one, rehabilitation has also improved, so that patients are able to find life worth living without a larynx.

Not only has the operative risk now been reduced to a minimum, but the period of hospitalization has likewise been greatly reduced. Not many years ago, the postoperative care of the laryngectomized patient involved a strenuous and complicated routine, requiring not only elaborate drainage which meant frequent dressings, but even wound irrigation at short intervals. Now, the use of antibiotics has eliminated all this, and wounds heal by first intention with pharyngeal fistula a rarity.

A simple one-stage excision is the usual procedure in all uncomplicated cases, whereas only a few years ago several of the leading operators advocated a two-stage operation. Whether a vertical or transverse T or U-shaped incision is used, and whether the larynx is removed from below upward or from above downward is a matter of the operator's individual preference, sometimes depending upon the location and extent of the tumor. The same may be said of the management of the hyiod bone. Some operators prefer simply to split it; others to remove the body, and still others to remove the bone in its entirety.

In a paper presented about ten years ago before the Eastern Section of this Society[13] we expressed preference for a basal anesthesia of avertin, supplemented by intravenous sodium pentothal, but for a number of years we have been using exclusively intratracheal anesthesia. Within the past year we have in a number of cases introduced the Woodbridge catheter through a tracheotomy incision in order to avoid the possibility of implanting tumor tissue in the subglottic larynx or the trachea, and, furthermore, as stated previously, to permit inspection of the interior of the larynx in the borderline cases, of the thyroid ala on the affected side. This procedure makes it easier to be sure of the completeness of removal of the tumor, and furthermore improves very much the prospects for voice development.

While we are speaking of conservative surgery of cancer of the larynx we should also mention the technique of partial horizontal laryngectomy, of Alonso,[9] which is suitable for limited supraglottic tumors.

Ordinarily we do partial laryngectomies under local anesthesia; but in borderline cases, where the excision must be fairly wide, and especially when we wish to make the final decision between partial and total laryngectomy on the operating table, we do first a tracheotomy under local anesthesia and insert a Woodbridge catheter through the tracheal incision. This leaves the larynx unobstructed by the intratracheal tube, permitting good inspection and palpation of the lesion and surrounding tissues; furthermore, this method affords equally satisfactory anesthesia for either wide partial or total laryngectomy.

Results of treatment by partial laryngectomy in 208 cases have recently been reported at the Fifth Pan-American Congress of Oto-Rhino-Laryngology, in San Juan[10]—88 per cent survival following primary treatment, with the salvage of eight patients who developed recurrence or metastasis, by second partial laryngectomy or other treatment, bringing the final five-year "cure" rate in this group of patients to 91 per cent.

One of the reasons it seems more important than ever to give the most careful attention to indications and techniques of conservative treatment is the fact that, as Ormerod[11] has stated, patients with cancer of the larynx are being seen and diagnosed at a much earlier stage than formerly, and hence these conservative techniques are applicable in a larger percentage of patients.

Total laryngectomy, like "laryngofissure," had a dismal and discouraging early history, with a high operative mortality and a high rate of recurrence. Gradually, however, with improved technique, the operative mortality rate has fallen to almost zero. In our clinic the only operative death occurring in the past 15 years, in 255 laryngectomies, was due to cardiac arrest, in a patient with complete heart block. While, of course, the operation must be admitted to be a disabling one, rehabilitation has also improved, so that patients are able to find life worth living without a larynx.

Not only has the operative risk now been reduced to a minimum, but the period of hospitalization has likewise been greatly reduced. Not many years ago, the postoperative care of the laryngectomized patient involved a strenuous and complicated routine, requiring not only elaborate drainage which meant frequent dressings, but even wound irrigation at short intervals. Now, the use of antibiotics has eliminated all this, and wounds heal by first intention with pharyngeal fistula a rarity.

A simple one-stage excision is the usual procedure in all uncomplicated cases, whereas only a few years ago several of the leading operators advocated a two-stage operation. Whether a vertical or transverse T or U-shaped incision is used, and whether the larynx is removed from below upward or from above downward is a matter of the operator's individual preference, sometimes depending upon the location and extent of the tumor. The same may be said of the management of the hyiod bone. Some operators prefer simply to split it; others to remove the body, and still others to remove the bone in its entirety.

In a paper presented about ten years ago before the Eastern Section of this Society[12] we expressed preference for a basal anesthesia of avertin, supplemented by intravenous sodium pentothal, but for a number of years we have been using exclusively intratracheal anesthesia. Within the past year we have in a number of cases introduced the Woodbridge catheter through a tracheotomy incision in order to avoid the possibility of implanting tumor tissue in the subglottic larynx or the trachea, and, furthermore, as stated previously, to permit inspection of the interior of the larynx in the borderline cases, where the final decision between a wide partial and a total laryngectomy is to be made on the operating table.

In the recent report already mentioned the percentage of five-year survival from primary treatment in 206 ("determinate") patients treated by total laryngectomy was 64 per cent, and the salvage of seven patients by further treatment raised this percentage to 67 per cent for the group initially treated by total laryngectomy. Combining the 208 ("determinate") patients treated initially by partial and the 206 treated initially by total laryngectomy, and taking into consideration the five-year salvage after treatment of recurrence or metastasis, the final five-year "cure" rate is 79.2 per cent for patients treated surgically. Incidentally, though this paper is concerned primarily with surgical treatment, it might be stated that during the same period 179 patients were treated by irradiation, with an initial five-year "cure" rate of 40 per cent, raised by "salvage" treatment of recurrence and metastasis to 47 per cent. Though Leroux-Robert and some others advise routine postoperative irradiation after laryngectomy, such therapy was not used in our series. The only patients who received irradiation after operation were patients who developed proven recurrence or metastasis. Of eight patients who had recurrence or metastasis after partial laryngectomy, subsequently treated by irradiation, only two survived five years, and of 27 developing recurrence or metastasis after total laryngectomy treated by irradiation only one survived five years. In recent years, surgical methods have been preferred in the treatment of recurrence and metastasis.

The most controversial point in connection with this subject at the present time concerns the management of the lymph nodes; *i.e.*, whether or not to do a simultaneous "evacuation" by *"en bloc"* dissection of the neck. If nodes are palpable, we feel that they should definitely be "evacuated"; if they are not palpable, the decision as to whether or not an "elective" or "prophylactic" neck dissection is indicated, should depend upon the location of the lesion. Since only 4 per cent of cordal tumors metastasize, and only 10 to 15 per cent of subglottic tumors do so, perhaps we are justified in awaiting the appearance of nodes in these cases, but in view of the fact that about 50 per cent of supraglottic tumors metastasize, we should probably consider simultaneous "neck dissection" at least on the side of the lesion, as relatively indicated. In the case of *lesions that are located anteriorly or bilaterally, it may be* difficult to decide upon which side to operate, and perhaps bilateral simultaneous "*en bloc*" dissection should be considered. According to the experience of Jorge Fairbanks Barbosa[13] bilateral operation can be done without serious complications if one internal jugular vein is preserved. We have done more than 50 unilateral simultaneous "*en bloc*" dissections, but these operations have all been done within the past five years, *so that it is not yet possible to give end-results of this procedure*

CONCLUSIONS.

The evolution of surgical technique in dealing with cancer of the larynx has been traced in the various modifications in the techniques of laryngofissure and other forms of partial laryngectomy as well as the techniques of total laryngectomy. The timely subject of indications for simultaneous unilateral or bilateral neck dissection is also considered. The authors' own end-results are summarized with the final percentage of 79.2 per cent five-year survivals following surgical treatment (328 out of 414 patients). Including all patients treated more than five years ago, by surgery and irradiation, the final percentage is 69 per cent (412 out of 593). The patients in whom simultaneous neck dissection was done do not figure in these statistics, since they were all operated upon within the last five years.

Finally, I should like to urge that all of us interested in carcinoma of the larynx and desirous of improving present methods and thus improving end-results, cooperate with the national and regional committees, and the International Committee for the Study of Cancer of the Larynx, in their efforts to standardize classification and nomenclature, as well as methods of reporting experience.

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SURGICAL TREATMENT OF CARCINOMA OF THE ANTERIOR COMMISSURE OF THE LARYNX.*

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Carcinoma involving the anterior commissure of the larynx or crossing over onto the anterior end of the opposite vocal cord even with unimpaired motility commonly recurs when treated by conservative surgery. By conservative surgery is meant any type of operative procedure less than laryngectomy, even though a segment of one or both thyroid alae together with portions of both vocal cords and an apparently adequate margin of surrounding normal tissue are removed. While it has been frequently stated by Clerf,[2] Schall[6] and others that lesions involving the anterior commissure are prone to recur when treated by thyrotomy, no figures could be found other than those of the indeterminate follow-up of LeJeune and Lynch[5] which were not based on five year results.

About 25 years ago the clipping operation for anterior commissure malignancy was advanced by Jackson[4] at which time he stated that if the method of subperichondrial dissection without cutting through the inner perichondrium was followed, the percentage of cures would be just as high as in cases in which the growth was limited to the central portion of the vocal cord. Satisfactory results by this type of surgical treatment should be secured when the lesion is confined to the interior of the larynx with no evidence of spread outside the larynx, and adequate normal tissue can be removed around the margins, yet when surgery more extensive than the original Jackson procedure was employed in a group of cases the recurrence rate was high. In the majority of cases of carcinoma

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involving the anterior commissure in which thyrotomy was employed, segments of the anterior portion of the thyroid cartilage on both sides were removed *en bloc* with the specimen. The internal perichondrium was not disturbed and the line of excision was through grossly normal tissue.

Broyles'[1] observations concerning the tendonous attachment of the anterior ends of the vocal cords to the thyroid cartilage and the absence of internal perichondrium in this locality, probably account for the many recurrences. On the other hand, extension into the anterior commissure may simply reflect the longer duration of the lesion and that the growth originated on one vocal cord with considerable time lapse before continuation into the anterior commissure, resulting in greater invasion of the tumor. In some instances carcinoma in the anterior commissure has extended through the cricothyroid membrane and thus into the strap muscles of the neck without demonstrable metastasis to the cervical lymph nodes.

TABLE I.

CARCINOMA OF ANTERIOR COMMISSURE OF LARYNX
(5 Year Follow-up Results)

	No. of Cases		5-Year Survivals	Determinate 5-Year Survival Rate	
Thyrotomy	42	75	26	62%	51%
Thyrotomy and further treatment	33		12	36%	
Laryngectomy	79	102	50	63%	55%
Laryngectomy and neck dissection	23		6	26%	
All cases	177		94		53%

A series of 259 cases of anterior commissure carcinoma operated upon at Jefferson Hospital on the service of Dr. Louis H. Clerf was reviewed, and the 177 cases followed five years or longer, form the basis of this report. Since 94 were alive and well the overall determinate five-year survival rate was 53 per cent (see Table I). The determinate five-year survival rate is based on the number of patients surviving five *years or longer in relation to the number of patients followed* for this length of time and forms the basis for an accurate appraisal of the results of treatment.

The indeterminate cases present a serious problem, but a reasonable evaluation is obtained when cases are considered in the light of five-year follow-ups rather than on the basis of *all cases treated, which include those which have not yet* reached five years and those who were totally lost to follow-up. There were 38 patients who died from causes other than carcinoma of the larynx in whom at the time of death there was no demonstrable evidence of local or distant cancer. Twenty-six patients were lost to five year follow-up (three years or more after surgery) who, when last seen, showed no evidence *of disease in the larynx. In the upper age group there were* eight patients over the age of 70 in whom the cause of death was not determined. All of these cases must necessarily be considered as failures in spite of the fact that no malignancy was demonstrable prior to death.

The histologic diagnosis of squamous cell carcinoma was confirmed in all cases, and in only 27 cases was the excised portion of the opposite vocal cord free from microscopic cancer. There were, in addition, 52 cases not included in this statistical study, with carcinoma of one vocal cord treated by thyrotomy in which the lesion extended up to the anterior commissure but did not involve this site.

Exact classification is essential to proper understanding of the results of treatment. In all of these cases the anterior commissure was invaded by cancer. In those in which thyrotomy was performed all of the other criteria for the operation were met. There was no evidence of metastasis to the neck, and mobility was unimpaired. In the group in which laryngectomy was performed the growth was much more extensive with fixation of one side of the larynx, spread into the ventricles, ventricular bands, or epiglottis and involvement of the opposite cord, while in a few subglottic extensions were also present.

Although statistics can be interpreted to suit one's liking, the important consideration in this group of cases is the number of recurrences in the cases treated by primary thyrotomy. Forty-four per cent of the thyrotomy group followed five years or longer had recurrence or metastasis. Some laryngologists contend that even if carcinoma recurs after thyrotomy, laryngectomy can still be done with a high survival rate. It was possible to salvage some of these at a later date by laryngectomy, but when the cases treated by primary thyrotomy and those salvaged by laryngectomy were considered together, the five-year survival rate was only 51 per cent (see Table II). To us recurrence means incomplete removal, and when carcinoma is found in the larynx, whether five, ten or fifteen years following the original growth, it is still a recurrence or continuation of the original growth. It is difficult to subscribe to the belief that a new cancer has developed. In looking back on the cases with recurrence, laryngectomy should have been the operation performed. Whereas 36 per cent of the group with recurrence treated later by laryngectomy lived five years or longer, when all the cases treated by thyrotomy plus those with recurrence and later treated by either laryngectomy or irradiation were considered, the five-year survival rate dropped to 46 per cent. Subsequent irradiation therapy in five patients with recurrence following thyrotomy was carried out with no five-year survivals. Additional surgical treatment to the larynx without laryngectomy was done in two of the cases after primary thyrotomy with no five-year survivals.

TABLE II.

CARCINOMA OF ANTERIOR COMMISSURE OF LARYNX
(5 Year Follow-up Results)

	Cases Followed	Survival 5 Years and Over	Recurrence Metastasis	Deaths Unrelated to Cancer
Thyrotomy	75	26	33	12
Recurrence and further surgery	33	12		4
		38 (51%)		16

Even though there is no evidence of local recurrence in the larynx, when the lymph nodes become involved, it is risky to do a block dissection of the neck for metastasis to the cervical lymph nodes without removing the original site of the lesion. While carcinoma may not be detected in the larynx, it probably exists, and unless the larynx is removed, together with the neck structures, the operation is inadequate. In the patients treated by primary thyrotomy there were six patients in whom cervical metastasis developed one to one-and-a-half years later, without evidence of local recurrence in the larynx, that were treated by radical neck dissection without removal of the larynx, and only one is alive four years following the original operation.

Considering that the five-year survival rate for all cases of carcinoma of the anterior commissure treated initially by thyrotomy is only 51 per cent, something further is needed to improve the results and bring them in line with those obtained by thyrotomy, when the lesion is limited entirely to one vocal cord, which approximates 75 per cent. A step in this direction can be taken if anterior commissure lesions are considered unsuitable for thyrotomy, and involvement of this area is used as a contraindication to this procedure. By this means it may be possible to eliminate the 44 per cent recurrence rate in those cases treated by thyrotomy, and the overall five-year survival rate should be greatly increased. At times, one is tempted to get by with conservative therapy when there is little evidence of extension to the opposite cord and the patient's desires in this matter may influence the physician's decision; however, when dealing with the life of the patient, this cannot be seriously considered, and percentage-wise the patient would have a better chance of living his normal life span after total laryngectomy.

In the group of 102 cases treated by laryngectomy the determinate five-year survival rate of those without metastasis was 63 per cent (see Table III). A much higher percentage of survivals was anticipated, but this included all cases with anterior commissure involvement regardless of the extensiveness of the lesion. While metastasis developed in 23 cases the salvage rate in these cases was only 26 per cent. The combined five-year survival rate for those patients treated by primary laryngectomy with simultaneous or subsequent radical neck dissection was 55 per cent.

If the figures were interpreted literally the conclusion might be reached that thyrotomy was the operation of choice because of the similarity of five-year survival rates. In cases without recurrence or metastasis primary thyrotomy yielded a 62 per cent five-year survival rate as compared with 63 per cent for primary laryngectomy, while the combination of the patients originally treated by thyrotomy and those subsequently treated by laryngectomy for recurrence gave 51 per cent five-year survivals in comparison with 55 per cent when they were treated by primary laryngectomy and subsequent neck dissection for cervical metastasis. It must be taken into consideration that those cases treated by thyrotomy had no impairment of mobility, no extension subglottically or onto the ventricular band while those submitted to laryngectomy had fixation of one side of the larynx with frequent involvement of adjacent portions of the larynx. The extent of the lesion is a prominent factor in the outcome of the case, and there was no question in those treated by laryngectomy but that total extirpation was necessary.

TABLE III.

CARCINOMA OF ANTERIOR COMMISSURE OF LARYNX
(5 Year Follow-up Results)

	Cases Followed	Survival 5 Years and Over	Metastasis	Deaths Unrelated to Cancer
Laryngectomy	102	50	23	20
Metastasis with neck dissection	23	6		2
		56 (55%)		22

No attempt has been made to compare the results of surgical treatment of carcinoma of the anterior commissure with primary irradiation therapy. The recurrences following thyrotomy which were treated by irradiation were all failures, and the patients did not survive. It has been demonstrated that cancer of the larynx can be treated successfully by irradiation and in the report by Harris, Silverstone and Kramer[3] the five-year survival rate was exceedingly high.

Our primary objective in carcinoma of the larynx is to increase the number of five-year survival rates whether this be by surgical means or irradiation therapy. When the surgical treatment of anterior commissure carcinoma consists of laryngectomy the disability is more marked, but more patients will survive five years when they are submitted to laryngectomy rather than thyrotomy.

SUMMARY.

In a series of 259 cases of carcinoma of the anterior commissure of the larynx treated surgically 177 cases were followed five years or longer with an overall determinate five-year survival rate of 53 per cent.

Of the 75 patients treated initially by thyrotomy without recurrence or metastasis the determinate five-year survival rate was 62 per cent, but 44 per cent of the 75 cases developed recurrence or metastasis. Some of the latter were salvaged later by laryngectomy, making the overall five-year survival rate for those cases treated by primary thyrotomy and those with recurrences requiring laryngectomy later 51 per cent.

In the group of 102 cases followed five years or longer without metastasis treated by primary laryngectomy the determinate five-year survival was 63 per cent even though the lesions were so extensive as to preclude a more conservative surgical procedure. The overall five-year survival rate for all cases including those treated by primary laryngectomy and those that developed cervical metastasis necessitating secondary radical neck dissection was 55 per cent.

While the similarity of five-year survival rates between those cases treated by thyrotomy and laryngectomy might be taken as an indication of the value of thyrotomy critical evaluation reveals that the cases treated by primary thyrotomy had a 44 per cent recurrence rate, of which only 36 per cent survived five years or longer.

The five-year survival rate of carcinoma of the anterior commissure of the larynx treated surgically can be increased if laryngectomy rather than thyrotomy is used as the primary procedure, and involvement of the anterior commissure is considered an absolute contraindication to the conservative operation.

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UNIVERSITY OF ILLINOIS, COLLEGE OF MEDICINE.

The next Laryngology and Bronchoesophagology Course to be given by the University of Illinois, College of Medicine, is scheduled for the period November 5 through November 17, 1956. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois, College of Medicine, 1853 W. Polk Street, Chicago 12, Ill.

INTRACRANIAL COMPLICATIONS OF OTITIC ORIGIN.[*][†][‡]

Part I. Role of Antibiotics on Acute and Chronic Aural Disorders.

Part II. Observations on Children Under Two Years of Age Among 50,000 Autopsies at the Los Angeles County Hospital.

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The spectacle of suppurative meningitis, septic thrombosis and brain abscess is still fresh in the memory of the clinical otologists of the preantibiotic era. Too often they stood helpless by the side of a moribund patient, witnessing the harvest of the Grim Reaper after a long and losing battle. The development and use of the new chemotherapeutic agents have revolutionized the modern practice of otolaryngology.

Sufficient time has now elapsed to enable one to assess both the merits and the limitations of these chemical agents. McLay[25] stated in 1954 that the best published results on otogenous meningitis before 1922 were those of Mygind,[26] who reported a recovery rate of 28 per cent. Watson[37] achieved a recovery rate of 84.6 per cent between 1944-1947. Blohmke and Link[3] of Frankfurt in 1951 reported that, with the use of antibiotics combined with the cisternal drainage through Trautmann's triangle, the mortality rate was reduced to 12 per cent.

Before the introduction of antibiotics, brain abscess claimed a toll of 50 per cent to nearly 100 per cent, depending upon the skill of the surgeon and the stage of the disease.

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[‡] The writer is indebted to Cyril B Courville, M D , the Director of the Cajal Laboratory, for the privilege of freely using the photographs and histological materials in his laboratory, and to the members of Pediatric Service for incorporating some of their observations

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Larroude,[21] of Lisbon, in 1950 recorded the mortality rate of brain abscess as 51.2 per cent. Natanzan[27] of Kharkov Clinic in 1951 accounted 18 cerebral abscess with 13 cures and eight cerebellar abscess with two cures, or a death rate of 43 per cent. Pennybacker[30] in 1950 stated that a death rate of brain abscess to be 29 per cent at the Radcliffe Infirmary, Oxford. Voyatcheck and Undrits,[36] in Russia, reported in 1953 the mortality rate to be 25 per cent. Ballantine and White[22] recorded in 1953 that mortality from brain abscess has been reduced at the Massachusetts General Hospital, Boston, from 80 per cent in the five-year period from 1936 through 1940, to 34 per cent from 1946 through 1950. Botterell and Drake[4] achieved a recovery rate of 86 per cent in 38 patients with localized encephalitis, brain abscess and subdural abscess, which had been treated with sulfonamides, or antibiotics, or both. Lateral sinus thrombosis has been practically wiped out.[27] Sepsis has been reduced from 25-40 per cent, to 4 per cent.[36]

Howard House,[17] in 1945, recorded that mortality from meningitis, in the Los Angeles County Hospital, was reduced from 89.4 per cent in 1935 to 14.3 per cent after sulfonamide compounds were used. The incidence of complications in the cases of acute otitis media was reduced to 20 per cent. The mortality rate of otitic complications was cut in half. Courville,[7] from the same institution, reported in 1955 that over a consecutive five-year period (1929-1933), prior to the introduction of sulfonamides and antibiotics, approximately 25 of every 1000 deaths were due to some intracranial complication of otitis media. During another five-year period (1949-1953), well within the antibiotic era, the mortality rate from the otogenous intracranial complications was reduced to 25 in 10,000 deaths, or one-tenth of the pre-antibiotic period.

It is proposed in this presentation to record the observations on the role played by the modern chemotherapeutic agents, over a 20-year period between 1935-1954 at the Los Angeles County General Hospital. Needless to say that the greatest single factor contributing to the reduction of the infectious diseases has been the advent of these potent drugs.

There are two parts in this paper: Part I deals with statistics on the otitic morbidity and mortality before and after the introduction of these chemotherapeutic agents. Part II presents significant altered aspects of the otitic intracranial complications in the antibiotic era, based on the clinico-pathological investigations among 50,000 autopsies at this 3,500 bed institution. It points to certain pitfalls and shortcomings as related to the practice of otolaryngology.

PART I.

Table I indicates (a) the total number of the patients discharged from 1935 to 1954; (b) incidence of deaths from all causes, and (c) mortality from acute and chronic ear disorders for this same period. Table II shows incidence of (a) acute otitis media and acute mastoiditis; (b) chronic otitis media and chronic mastoiditis; (c) number of patients having simple or cortical mastoidectomies, and (d) those who had either a modified or complete radical mastoidectomy for relief of chronically draining ears and chronic mastoiditis. The comparison of the incidence of acute and chronic otitis media and their complications among both adults and children, before and after the use of antibiotics, is illuminating. The highest incidence of acute otitis media and acute mastoiditis in the preantibiotic era was in 1936. Then there were 589 patients among 62,129 admitted, or one in 105. Following the introduction of sulfa drugs and antibiotic substances, there was a dramatic decline in acute otogenous morbidity for seven years, 1937-1943. The lowest incidence was recorded in the fiscal year 1943, when there were 153 among 54,508 patients, or one in 356. Since 1944 there has been a spectacular rise in the incidence of acute otitis media. The largest number of patients with acute otitis media in a single year was in 1949, when there were 918 among 84,254 patients. This is a ratio of one to 90 patients. This high incidence of acute otitis media may possibly be attributable to a combination of several factors: 1. An increased proportion of children under 10 years of age; 2. A relative increase in benign non-

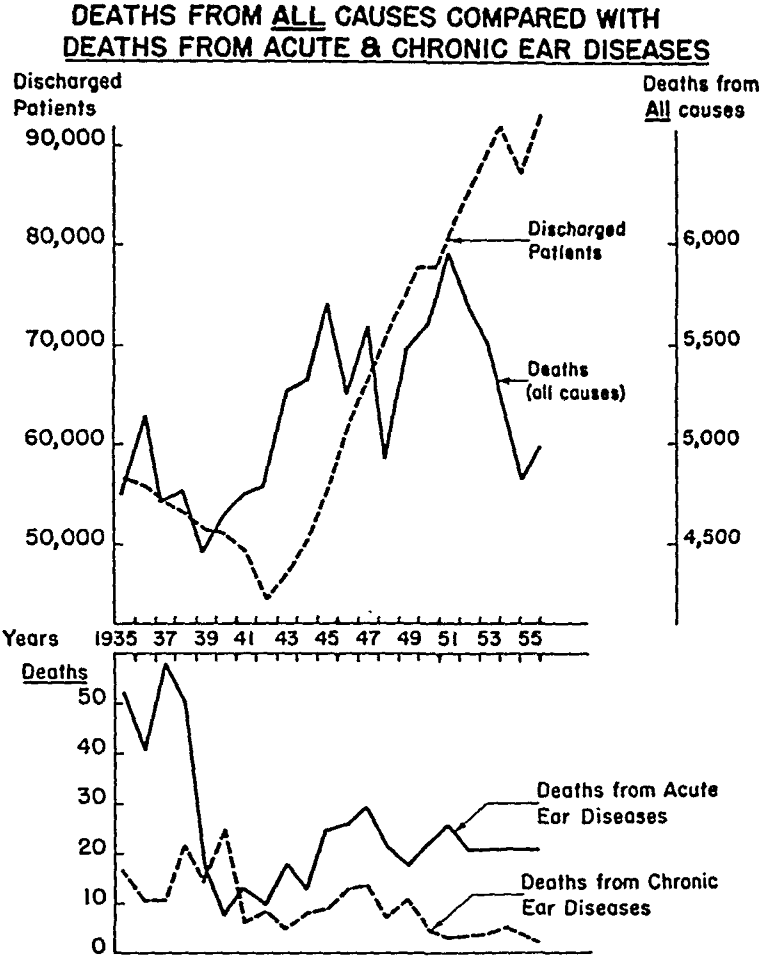

Table I

suppurative otitis; *3.* The alteration of bacterial flora as the result of extensive use of antibiotics; *4.* To as yet undetermined causes. There has been an influx of general population, notably among children under 10 years of age who are highly susceptible to acute upper respiratory infections. The relative increase in non-suppurative otitis media may be due to the increasing incidence of minor as well as major allergic response to drugs.

As a result of extensive use of the antibiotics, many of the

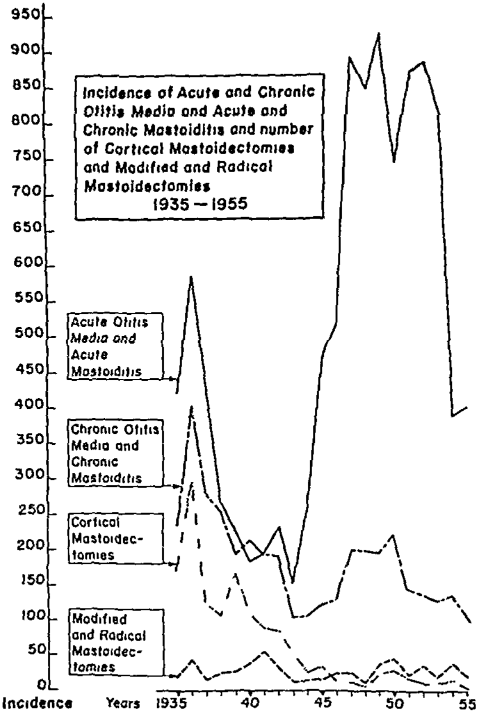

Table II

more highly virulent strains of bacteria responsible for these infections have been eliminated from the picture. There remains, however, drug resistant strains of these organisms. In any event the virulence of the organisms responsible for the cases in recent years has apparently been greatly reduced, as reflected by the lessened number of surgical mastoiditis. The greatest number of simple mastoidectomies were performed in 1936, when there were 290 operations among 62,129 patients admitted that year. Since the introduction of penicillin and other antibiotic agencies, there has been a decline in the number of patients with acute mastoiditis who required a cortical operation. In 1955 there were only three among 98,147 patients upon whom a cortical mastoidectomy was performed. This is the lowest incidence of this surgical procedure in the history of the Los Angeles County Hospital. The incidence of chronic otitis media followed a similar pattern of rise and fall as in the acute cases. The number of either modified or radical mastoidectomies ran closely parallel to those of cortical mastoidectomies. Since 1946 there have been a larger number of radical mastoidectomies performed than the cortical mastoidectomies.

PART II.

Spectacular as has been the role played by antibiotics in the reduction of morbidity and mortality from infectious diseases, there has been a universal recognition of an increased incidence of undesirable features in the use of these drugs.[10,12,16,19,20,28,29,31,33,34,35] Among several factors influencing the antibacterial therapy, mention should be made of *1*. The development of antibiotic-resistant strains of micro-organisms; *2*. emergence of super infection due to alteration of normal human bacterial flora; *3*. hyper-sensitivity of the host; *4*. untoward side effects and toxicity; *5*. chronicity and recurrent infection following inadequate administration; *6*. unpredictable synergism and possible antagonism when two or several drugs are combined; finally *7*. one of the serious disadvantages in the antibiotic therapy is the increasing tendency of masking symptoms in clinical otology. A significant number of deaths attributed to intracranial complications continues, particularly among children under two years of age.[13] At the best their otoscopic examinations are often difficult. After treatment with antibiotics, the drum membrane appears gray, thickened and sometimes even retracted, due to lessened intratympanic pressure. The exudate is usually thick, viscid, and ropy, not favoring free drainage.

In this presentation the discussion is limited to this age group under two years. These were all treated at the Los

Angeles County Hospital since 1944, well after the introduction of antibiotics, and in each case the essential lesion was verified by autopsy. Excluding two children, who died from septicemia, there were 16 cases in which death resulted from intracranial complications. Each received varying quantities of antibiotics before admission. In almost every instance the initial symptoms of convulsions, high fever, and gastrointestinal disorders over-shadowed the presence of the infection in the middle ear; thus, the acute otitis media was either unsuspected or recognized only very late in the course of the disease. The usual complications of thrombosis of the venous channels, suppurative meningitis and brain abscess in this series were found.

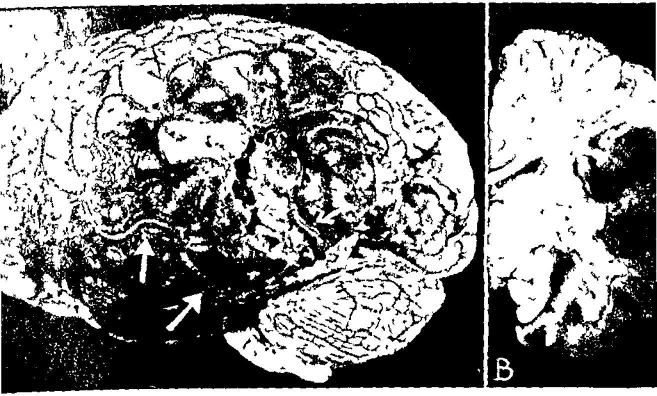

Fig 1 Case 1 Otitic thrombosis of the inferior anastomotic and inferior cerebral vein, with subarachnoid hemorrhage and red softening of the underlying cerebral cortex

VENOUS COMPLICATIONS.

Two types of thrombosis of the dural sinuses are now recognized. They are *1.* septic or infectious thrombosis; *2.* marantic or primary thrombosis. To the older generation of otologists the clinical picture of lateral sinus thrombosis and bacteremia in the presence of acute coalescent mastoiditis with recurrent chills, swinging temperature and convulsions in children, in the pre-antibiotic age, is all too familiar. Marantic thrombosis develops from alteration in the quantity and quality of the blood and from reduction in the speed of flow secondary to dehydration, inanition and lack of normal activity. Marantic thrombosis involves predominantly the superior longitudinal sinus or straight sinus. Other sinuses are at times affected. The occlusion of the venous channels brings about edema, multiple petechial hemorrhages, softening of the basal ganglia with high mortality. In the present series there were four cases of extensive thrombotic process in the cerebral venous system. In one instance both cavernous sinuses were thrombosed.

Fig 2 Case 2 Thrombosis of the superior cerebral veins incident to otitic thrombosis of the superior longitudinal sinus Red softening of the white centrum of both cerebral hemispheres

Case 1 J. S. (L.A.C.H. No 1037-668); a 14-months old Mexican male was admitted with the history of upper respiratory infection and diarrhea of four days' duration. He was markedly dehydrated, acidotic and his eardrums were dull. The temperature on admission was 104°, the white blood count was 39,800 per cu mm, with polymorphonuclears of 82 per cent He was placed on the program of hydration, penicillin, sulfonamide and whole blood transfusion. His course, however, was downward, and he expired 48 hours after admission.

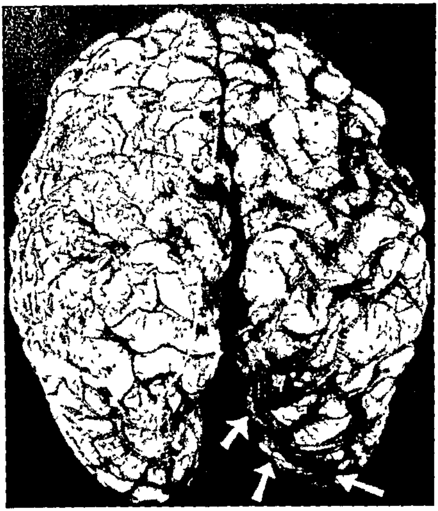

Fig. 3 Case 3. Otitic thrombosis of the right lateral and the superior longitudinal sinuses with secondary involvement of the superior cerebral veins; none evident over the posterior portion of the right cerebral hemispheres (arrows).

At necropsy examination (No 38-429) there was found a suppurative right otitis media and mastoiditis, and an extensive venous thrombosis affecting the middle portion of the left cerebral hemisphere. There was also a softening of the left temporal and parietal lobe (see Fig. 1).

Case 2: M. T. (L.A.C.H. No. 1009-643), a well developed and well nourished Caucasian male infant, two months of age, had diarrhea and vomiting after each feeding for a period of three days. He had six watery stools on the day of admission. Both of his eardrums were injected and full. The child continued to have diarrhea with a low grade fever and mild leucocytosis. He was placed on penicillin, sulfonamide, plasma transfusion and forced hydration. His condition became gradually worse and progressively lethargic, and he expired two weeks after admission to the hospital.

On autopsy (36953) there was found greatly distended and thrombosed superficial vessels of the meninges. There was also a complete thrombosis of the sagittal sinus, together with the superior cerebral veins, both lateral sinuses and cavernous sinuses. A mild hemorrhage had taken place over the right cerebral hemisphere and to a lesser extent over the left cerebral hemisphere. There was an extensive red softening of the white substance of both hemispheres (see Fig. 2). Staphylococcus coagulase positive was recovered from both middle ears which contained thick mucopurulent exudate.

SUPPURATIVE MENINGITIS.

Suppurative meningitis was the most frequent of the various lethal complications occurring in this series. There were ten instances of meningitis, including three cases due to pneumococcus, one each of hemophilus influenza, beta hemolytic streptococcus and meningococcus. In four instances the organism failed to grow on culture media, perhaps because their viability was severely attenuated under the influence of chemotherapy, in spite of the fact that their spinal fluid was opaque to ground glass in appearance. Infection in these cases usually develops by retrograde thrombophlebitis from the focus in the middle ear. Less frequently it may occur as an adjacent penetration from the dural layer.

There are several reasons for the failure of antibiotics in the treatment of suppurative meningitis. In the first place, the anatomical structure of the subarachnoid space permits a wide diffusion of the death-dealing organism and provides a large area for a rapid absorption of end product of the invading bacteria; second, the various antibiotic agents are incapable of rapidly penetrating the thick exudate to complete its dissolution; third, the lack of natural immunity in the debilitated and marasmic child is probably the deciding factor that tips the scale unfavorably. Histologic examination of the autopsy material showed that where blood-borne antibiotic agencies come in immediate contact with the exudate in the subarachnoid space it first becomes thin and then disappears, resulting in the establishment of bacteria-free zones. In favorable cases the dissolution of the exudate goes on progressively. Isolated patches are likewise dissolved, and the meninges become clear. In the presence of overwhelming infection, or when the inadequate or inappropriate chemotherapy is given, death occurs after an acute or subacute course. In the first instance, a thin exudate is found at autopsy often associated with focal subarachnoid hemorrhages. To elucidate these points, the following cases are cited:

Case 8: C. B. (L.A.C.H. No. 1286 912), an eight-months-old Negro girl became irritable and feverish two days before admission to the hospital. She developed a cough and running nose. On admission, she had a temperature of 105° and was lethargic. Her right eardrum was injected. Diagnosis of meningitis was made. She was given penicillin, terramycin and sulfa drugs, but failed to respond. There were numerous convulsive seizures, and the child died after 18 days in the hospital and three weeks after the onset of the symptoms, from what appeared to be respiratory failure.

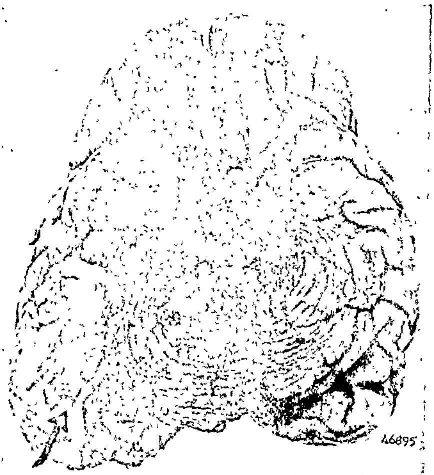

Fig. 4. Case 8. Subarachnoid hemorrhage and diffuse meningitis associated with softening of the interior of the right temporal lobes (arrow) in a case of otitis media in an infant.

On autopsy (No. 46-895) the meninges were found to be somewhat opaque and the purulent exudate was most abundant over the left frontal lobe. There was a massive meningeal hemorrhage filling the basilar cisterns and almost completely enveloping the pons and medulla (see

Fig. 4). The interior of the right temporal lobe was softened. Both middle ears contained pus from which pneumococcus were isolated.

Case 10: R. M. (L.A.C.H. No. 1350-317), an acutely ill, six-months-old Mexican female infant was admitted to the hospital with a diagnosis of suppurative meningitis. At three months of age the child was treated at this hospital for diarrhea and vomiting. Four days before the present admission she had chills and vomiting. For two days the child had been coughing, with a temperature of 105°. Tympanic membranes were bulging. Myringotomy yielded purulent material from both ears, which on culture indicated the presence of a pneumococcus; the blood culture was also positive for this organism. A vigorous antibiotic regimen with supportive measure was instituted; however, she had numerous convulsions and expired the following day.

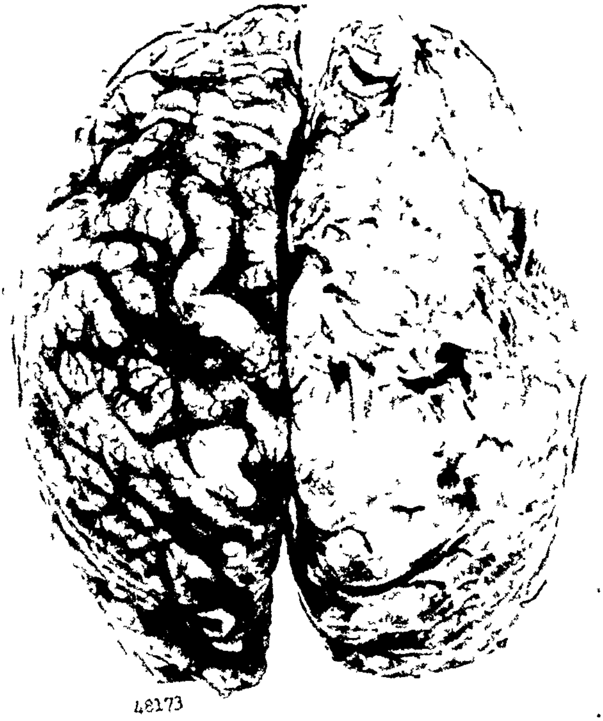

Fig. 5. Case 10. Advanced purulent otitic meningitis, chiefly right hemispheral.

On autopsy (No. 48-173) the meninges showed marked injection. They were opaque over the dorsolateral surface of the right cerebral hemisphere, being filled with a grayish-green purulent exudate. There was a generalized edema of the brain. Histologic examination of the medulla indicated an early acute toxic nerve cell change with pyknosis (see Fig.

7) The right middle ear and mastoid contained a large amount of olive green purulent material The left contained only a minimum amount.

ACUTE ENCEPHALITIS AND BRAIN ABSCESS.

In pre-antibiotic days the most frequent form of otitic brain abscess was secondary to chronic otitis media and mastoiditis, often complicated with the presence of a cholesteatoma. The abscess usually develops as a consequence of the infection of the adjacent dural layer with subsequent spread either through venules or a chronic osteomyelitis. In acute otitis media and acute coalescent mastoiditis, particularly in children and adolescents, the distant lesions occur probably from a retrograde thrombophlebitis. The regional venous channels are less frequently involved. At times an abscess develops from the blood stream infection.

In the present series, there were two instances of regional encephalitis with an early central inflammation. The first case was secondary to massive thrombosis of the cerebral veins. The second one developed with recurring meningitis and unoperated petrositis. The report of these two cases follows:

Case 15· E B (L A C H. No 1012 831), a well developed but poorly nourished five-weeks old Mexican female infant, had diarrhea and vomiting for four days prior to admission to the hospital. Her temperature on admission was 105°. The pulse was 180 per minute She weighed seven pounds Both of her eardrums were grossly injected. A myringotomy yielded an alpha streptococcus She received penicillin, sulfonamides and hydration After a stay in the hospital for a period of two weeks she expired from respiratory failure

On autopsy (No 36 974) there was found a bilateral thrombosis of the cerebral veins of the frontal and temporal regions, associated with regional subarachnoid hemorrhage and acute red softening of the underlying brain with secondary supperative encephalitis Both ears showed suppuration

Case 16 G G (L A C H. No 1374 591), an 18 months old white male child was admitted to the hospital with a history of diarrhea for four or five days followed by a left otorrhea He had finally developed signs of meningitis Three months prior to his admission to another hospital, the child fell on his head. An electro encephalogram there had revealed an abnormal left parieto occipital focus Right sided convulsions, a left lateral rectus paralysis, a contracted left pupil, and choked disc were observed at this time A persistent serous drainage from the left ear was noted He was placed under heavy antibiotic therapy in that hospital After six weeks the otorrhea ceased spontaneously, and the child appeared to be on the road to recovery Three days before admission to the Los Angeles County Hospital the child again fell and struck his head He began to vomit and developed a spiked temperature Purulent otorrhea then recurred from the left ear. Despite massive antibiotic therapy and a left myringotomy the child expired the following day. An X-ray taken two days before his death disclosed an acute left mastoiditis and petrositis.

At necropsy (No. 48-185) a heavy collection of pus was found over the tip of the left temporal lobe. The lateral ventricles were reduced in size because of cerebral edema. The left temporal lobe showed regional encephalitis with early central inflammation, as observed in the first stage of abscess formation. The clinical behavior of this child reminds the otologist of the pre-antibiotic era, a mastoiditis due to pneumococci Type III, with its insidious onset, a slow but steady progress, a treacherous and temporary abatement of major symptoms under medical or surgical therapy, and a final dramatic explosion, resulting in a fatal issue.

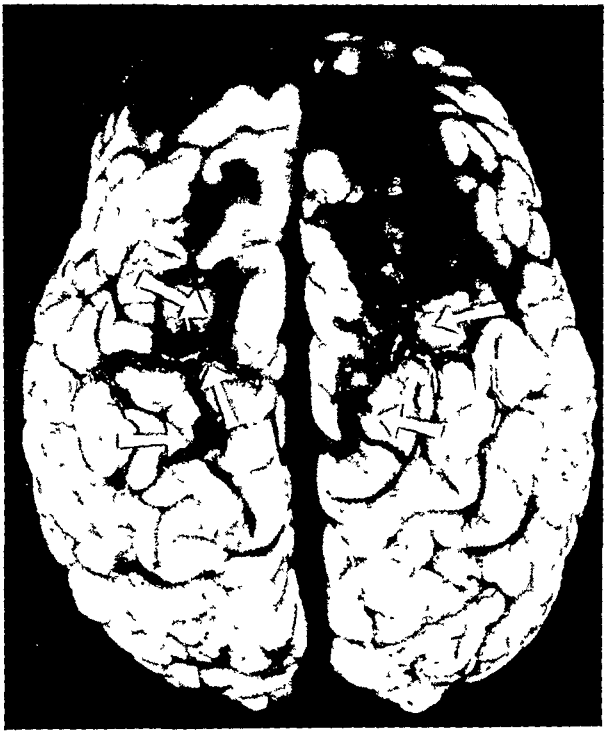

Fig. 6. Case 15. Subarachnoid hemorrhage secondary to retrograde otitic thrombosis of superficial cerebral veins (arrows)

GENERAL DISCUSSION.

It is of particular interest that in this series of cases the symptoms of U. R. I. were first observed in five; and an episode of initial diarrhea, vomiting and high fever had occurred in eight patients. In three cases upper respiratory symptoms were also present in addition to diarrhea. In the past, many conflicting views have been expressed among American otologists and pediatricians[1,6,11,15,22,23,32] as to the relation of acute otitis media and gastrointestinal disorders among children. A seemingly unending controversy followed until Wishart's[38] investigation in 1930 finally appeared to settle the issue. The evidence here strongly suggests that debilitated and dehydrated children whose vitality was lowered, due to the altered electrolytes and inadequate immunity, were unable to cope successfully with subsequent or co-existing infection of their middle ears. It was recorded in a number of these instances that the symptoms abated during the administration of penicillin, but recurred when it was discontinued or given in inadequate doses. Penicillin and sulfa drugs often masked signs of grave complications.

Almost every one of these children had been first seen by general practitioner or pediatrician, and all received varying amounts of penicillin. There is a widespread notion among some practitioners of the healing art that all that is required in the treatment of infectious diseases of the ear is the administration of penicillin and other supposed-to-be-cure-all drugs. They have developed such a complacent attitude that they often omit a thorough otoscopic examination. Myringotomy has almost become a lost art.

All of these children were first admitted to the Pediatric service of the hospital. Only two of them had draining ears. Diarrhea, vomiting, dehydration, hyperpyrexia, lethargy, convulsions and signs of grave respiratory disturbances were so marked that the presence of otitis media was unsuspected in seven cases, either at the time of their admission or during their hospitalization. In four cases the tympanic membrane was recorded to be full and bulging, but no mention was made of attempts at surgical drainage. In four cases a myringotomy was performed; however, all of these 16 children at autopsy were found to have purulent exudate in one or both of their ears.

A study of this series of fatal cases of the otitic complications has brought to light some indisputable facts. Since almost every child in this series was first seen by a general practitioner or a pediatrician, the problem of prevention is one which obviously lies in their diagnostic judgment. Promiscuous use of antibiotics, often in too small quantities, can not be too strongly condemned. The choice of the drug must be based on the behavior of the bacteria as revealed by sensitivity tests. Meningitis still proves to be the chief lethal complication secondary to fulminating acute otitis media in the poorly nourished and dehydrated infant. We must all be alert in recognizing the earliest signs of meningeal irritation. To wait for a spontaneous rupture of the tympanic membrane is only to court serious intracranial complications. It is a surgical axiom that where pus occurs it must be drained. This principle remains unchanged and unchangeable even after the introduction of modern antibiotic agents.[5,6,20]

SUMMARY AND CONCLUSION.

1. Acute and chronic middle ear infections and their intracranial complications that had been observed over a 20-year period, before and after the introduction of sulfa drugs and antibiotics in a 3,500-bed general hospital, is presented.

2. For seven years (1937-1943) immediately after the institution of sulfa and antibiotic therapy, the incidence of acute otitis media was reduced from 1 in 105 to 1 in 260 patients. There has been a corresponding decrease in the number of acute surgical mastoiditis. During the past decade (1945-1954) almost as many cases of acute otitis media (one in 115) as in the pre-antibiotic era were observed. This increase was attributed to the alteration of bacterial flora coincident to antibiotic therapy, to a possible increase in number of serous otitis, and a larger proportion of children under 10 among the general population.

3. One of the serious disadvantages of antibiotic therapy appears to be the masking of symptoms, particularly among children under two years of age, often leading to fatal intracranial complications.

4. Altered aspects of the tissue response after the administration of inadequate and inappropriate chemotherapy is presented from the pathological investigations on four cases of cerebral venous thrombosis, ten of suppurative meningitis, and of acute encephalitis among 50,000 autopsies between 1918 and 1954.

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SYMPOSIUM ON OCCUPATIONAL NOISE.

A special one-day conference Monday, September 17, 1956, will be presented by Yale University School of Medicine, Department of Surgery, Section of Otolaryngology, Department of Public Health, Section of Occupational Health and Grace-New Haven Community Hospital Hearing and Speech Center in cooperation with Liberty Mutual Insurance Company.

This one-day conference has been planned for industrial executives, physicians, safety engineers, and others who are concerned with problems of occupational noise.

Location: Fitkin Amphitheater, Yale-New Haven Medical Center, 789 Howard Avenue, New Haven, Connecticut. Date: Monday, September 17, 1956. Registration at 9:30 a. m. Fee: $15.00 per person (includes luncheon). Total enrollment will be limited in number. Only those who have registered in advance will be admitted. Further information may be obtained by writing to the Assistant Dean of Postgraduate Medical Education, Yale University School of Medicine, 333 Cedar Street, New Haven 11, Connecticut.

TONSILLECTOMY: TWO MILLENNIA OF HEMORRHAGE AND CONTROVERSY.*†

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Atlanta, Ga.

Although the operation in point was liable to resolve itself into physical combat between the surgeon and his patient, it is one of the oldest surgical procedures. According to Celsus,[1] a Roman aristocrat who lived during the time of Christ, removal of tonsils was considered an ordinary procedure in ancient Rome. He directed that when the tonsils had become indurated after inflammations, "they ought to be disengaged all 'round by the finger, and removed; if they are not separated by this method, it will be necessary to take them up with a blunt hook, and separate them with a scalpel; then to wash the ulcer with vinegar, and anoint the wound with a styptic application." The description of this technique with such casual reference to the control of bleeding convinces me that Celsus was the first man in history about whom it could be said that he either did not tell the entire truth or did not take out tonsils himself.

One of the earliest surgeons to profess publicly his concern with hemorrhage after tonsil surgery was Aetius of Amida. Aetius, who practiced in Constantinople in the Sixth Century, A. D., compiled what is said to be the best description of diseases of the ear, nose and throat in the literature of antiquity.[2] He was afraid to take out the whole tonsil because of the danger of hemorrhage if the gland were incised too deeply; so, after pulling the tonsil forward with a hook, he cut off only the most prominent portion.

In the next century, however, Paul of Aegina[3] described complete tonsillectomy clearly and precisely, discussing in de-

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tail the prevention and treatment of post-operative bleeding. In fact, 1200 years were to pass before we find recorded again (in the 19th century) a technique as refined. Unfortunately, shortly after the time of Paul, Europe descended into the Dark Ages and, along with the general disintegration of knowledge, tonsillectomy apparently fell into disuse. When we come to the time of the School at Salerno, which was the Johns Hopkins of the Middle Ages, we find tonsil surgery limited to lancing of peritonsillar abscess.[4]

During this time the medical knowledge of the Greeks and Romans passed into the hands of the Moslems, who kept it alive until the rebirth of learning, centuries later, in Europe. Albucasis,[5] known as the most daring and brutal of the Arabian surgeons, while he removed tonsils in one way or another, greatly feared hemorrhage and did not advise the operation unless the tonsil "be of white color, and round, and the root of it, besides, small. For if the basis be very large, a flux of blood . . . is much to be feared: which has often indeed happen'd in this case, so as to prove extremely troublesome, if not dangerous."[6]

With the coming of the Renaissance, medicine on a more enlightened basis returned to Europe. Tonsillectomy, however, was slow to regain its previous acceptance and degree of development. Removal by means of the scalpel, as had been previously dscribed, was seldom undertaken because of the bleeding which was described as always violent and sometimes mortal;[7] therefore some of the more ingenious surgeons of the time tried to eradicate them by means other than cutting. The application of red hot cautery or corrosive chemicals was tried rather extensively[8,9] but needless to say did not attain much popularity with the patient. Another method involved the use of the ligature, which was developed by the great Guillemeau, whose book incidentally is a treasured collectors' item.[10,11] The tonsil was grasped and pulled out of its bed and then a noose of thread or wire was slipped around the base and tightened until the circulation was cut off. The ligature being left in place, the tonsil became gangrenous and in four or five days fell away. This method, however, was frequently accompanied by severe infection, not to speak of intense and prolonged pain.

Wiseman, surgeon to King Charles II, was brave enough to cut off the tonsil with scissors as soon as the ligature had been tightened and the blood supply stopped.

He, too, had problems, as can be seen from the following case which he described in 1686. "A Person of Honor, aged 10 years, having been some years diseased with preternatural swellings in her Tonsils . . . was brought to London . . . for Extirpation of them. . . . I placed her in a clear light, her head leaning to a bed post, Mr. Arris sitting behind holding it steady. . . . Having divided the one Tonsil, and diminished the other (by means of a long-pointed caustick stone fixed to a stick and with a Firre-stick dipt in oleo vitrioli), I caused an instrument to . . . convey a ligature about the basis of them. . . . Holding the ligature . . . with my right hand, I passed a pair of Probe-scissors . . . close by the said basis and . . . clipt . . . off the bigger part of it . . . at which instant she choaked. The Ladies seeing it, shrieked, and became troublesome . . . I desired Mr. Arris to sit still while the patient choaked. . . . She gaping I passed into her Throat the Instrument I had made the Ligature by, and raising up the Excrescence, she recovered, and cried, 'Shall I live?' I replied, 'Yes, if you sit still.' . . . But being now better informed . . . I passed my crooked probe scissors down lower, and cut it clean off by the roots, and brought it away with my Ligature. . . ."[12]

During this time it was believed that the secretions of the nose were formed in the brain and entered the nasal cavity through the cribriform plate of the ethmoid, and that the purpose of the tonsils was to absorb these secretions and send them back again. This belief goes back to the time of Hippocrates.[13] Galen,[14] called the Greek Dictator of Medicine for a thousand years, also had previously cautioned against cutting off too much of the gland for fear of injuring the voice and making the patient liable to inflammations of the lungs.

So. during this period, many physicians were opposed to removal of the tonsils for any reason, and agreed with Dionis, one of Europe's most prominent surgeons around 1700, who wrote: "Some of our ancestors propose the separation and evulsion of these glands, which operation they very easily perform'd, and assure us that it will never incommode the patient: I refer you to the methods which they propose of doing it, which I think very cruel; and would be glad of some better security for their success than their bare word; for the function of these glands being to separate and filtrate the secrosities which serve to moisten the tongue, larynx and esophagus, these parts must find themselves deprived of that dew, which is of great use in the tempering of the air in the lungs, and sliding the nourishment into the stomach."[15] Although in the minority, a more courageous French surgeon maintained that: "l'o'peration necessaire n'est ni dangereuse ni difficile."[16]

The era of wholesale tonsillectomy began even before the discovery of anesthesia, when in 1828 Philip Physick,[17] professor of surgery at Pennsylvania, devised the first practical tonsillotome. He reported its use in four cases wherein the operation took only a moment, causing very little pain and bleeding. This technique was eagerly taken up by his American and Continental colleagues, many of whom modified the instrument slightly and extolled theirs as more foolproof than the others, especially with regard to hemorrhage.[18] The discovery of the significance of adenoids by Meyer[19] of Copenhagen, in 1868, only added fuel to the flames. The race was now on and by the beginning of the 20th Century a tidal wave of articles on "T's and A's" was engulfing the literature. Although many of these articles contained fervent descriptions of the author's own bloodless technique, by 1921 the problem of hemorrhage was such that the entire program of the meeting of the Section on Oto-rhino-laryngology of the British Medical Association that year was devoted to a symposium of eleven papers, all on the causes, prevention and treatment of hemorrhage in connection with tonsillectomy.[20] In 1926 Jay[21] was prompted to write: ". . . it is certain that in no other operation is considerable hemorrhage viewed with so much equanimity by the surgeon."

Despite this long heritage, with consequent reluctance of many patients to have this operation performed, it is still the mainstay of the specialist's budget. It is not surprising, however, that hemorrhage can still be somewhat of a problem. In the words of Poe: "Vainly had I tried to borrow from my books surcease of sorrow" for in the past five years, I have had eleven cases of post-operative hemorrhage following adenotonsillectomy in children. The bleeding in these cases was arterial in nature, moderately profuse, and occurred after the operation as follows: seven patients bled from the adenoidal fossa within four hours; two others bled from a tonsillar fossa also within four hours; the tenth patient bled from a tonsillar fossa two days later. The eleventh case, a physician's daughter, bled three hours following the procedure, again on the seventh postoperative day and yet again on the tenth postoperative day, each time from the right tonsillar fossa.

While representing barely 1 per cent of the total cases operated upon during this period, and although at no time was there danger of anyone bleeding to death, these eleven children caused me no little anxiety. The readministration of an anesthetic to a partially exsanguinated child whose airway is obstructed by blood can be one of the grimmest undertakings in all surgery. Even under local or no anesthesia, the customary procedures are apt to be harrowing, both to the patient and the surgeon.

The principal treatment given these children consisted of blood transfusion, this therapy successfully reducing the need for the measures ordinarily used. A surprising benefit was an apparent hemostatic effect. In all cases but two in this series the bleeding stopped before the transfusion was completed and without recourse to any other manipulation or instrumentation. As to the exceptions: it became necessary in one instance to crush a bleeding vessel in a tonsillar fossa under local anesthesia; in the other instance a postnasal plug was inserted after the child had reacted from the anesthetic.

The chief advantage of this treatment is, of course, the replacement of blood loss; however, it is often difficult to calculate accurately the amount lost. In a small child, especially, the relative blood loss is easily misjudged and on more than one occasion, prior to this series, when it seemed that a child had lost only a small amount of blood, a surprising degree of secondary anemia was later found to be present. In this series, after the bleeding had stopped, return of the pulse rate to normal was found to be a useful clinical indication that the blood had been replaced, since subsequent blood counts showed no anemia.

On the other hand, the possibility of giving too much blood with consequent reactivation of the bleeding should not be excluded. In the first excepted case the bleeding had actually stopped during the transfusion; however, the blood was continued after the calculated amount lost had been replaced. After some 200 cc. of excess blood had been given, the bleeding recurred, and it was then that the vessel was crushed. One week later this child's hemoglobin determination was a gram higher than it was preoperatively.

In using this method of treatment, consideration was given to the conservation of veins and elimination of as many venipunctures as possible. Multiple venipunctures not only deplete the limited number of readily accessible veins, but may render the child less cooperative because of added pain and terror which sometimes causes an increase in the rate of bleeding. The single venipuncture method was, therefore, used.

In this procedure, as soon as it becomes apparent that a child is bleeding postoperatively, a good arm vein is selected and an arm board is put securely in place. A new 19 or 20 gauge needle is used to withdraw the blood for cross-matching. The needle is left in the vein and a saline infusion is started through it. Preparation is made to facilitate the vomiting of blood so that the needle will not be dislodged should this occur. When the blood for transfusion is ready, it is then given through the same needle.

Strangely, this treatment is not nearly so effective in adults as in children. In the series just presented, the children ranged in age from four to ten years of age. During the same period transfusion therapy was used in the five adult patients who bled postoperatively, their ages ranging from 18 to 36 years of age. This treatment controlled the bleeding in only two cases. In the remaining three cases it was necessary to ligate a "spurter" in a tonsillar fossa.

With the exception of a mild, fleeting, generalized rash which occurred in the case of the physician's daughter, no allergic reactions followed the use of this therapy in any age group.

SUMMARY.

1. The controversy over whether or not to take out tonsils had been going on for at least 2,000 years. The two principal factors have pertained to the danger of hemorrhage and the protective function of the tonsil.

2. The treatment of choice for moderate arterial hemorrhage following childhood adenotonsillectomy is found to be blood transfusion. This treatment exerts an apparent hemostatic effect in addition to replacing blood loss. The hemostatic effect is less in adults than in children.

EPILOGUE.

While this treatment is of value, I realize it is by no means the solution to the problem. Since all this trouble began in the early Christian era, it is only right that the nearest thing yet to the solution should also come from this time. This, the answer to an otolaryngologist's prayer, is found in Revelations, 20th Chapter, the first three verses:

> And I saw an angel come down from heaven, having the key of the bottomless pit and a great chain in his hand.
>
> And he laid hold on the dragon, that old serpent, which is the Devil, and Satan, and bound him a thousand years.
>
> And cast him into the bottomless pit, and shut him up, and set a seal upon him, that he should deceive the nations no more, till the thousand years should be fulfilled: and after that he must be loosed a little season.

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THEORY AND PRACTICE OF TYMPANOPLASTY.

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Today if we are faced with the problems of functional surgery of the middle ear in chronic otitis media, we are, thanks to modern audiology, equipped with a thorough theoretical foundation. Typical defects of the sound conducting apparatus of the middle ear create a hearing loss with a conductive component, *i.e.*, with a gap between bone conduction and air conduction in the threshold audiogram measuring from 40 to 60 dbs. If the hearing loss for air conduction markedly exceeds 60 dbs., the suspicion arises that there is a simultaneous damage to the sound perception apparatus of the cochlea present.

The various types of hearing losses are known not only from clinical experience, but have also been measured in a typical manner with the cochlear potential in animal experiments (Wever, Lawrence, Ranke, Gisselson, Wagemann, Golo).

The tympanic membrane has two functions, sound pressure transformation for the oval window and sound protection of the round window. The greatest hearing loss will be found if both labyrinthine windows are subject to sound protection; this occurs if the drum is intact but the ossicular chain interrupted (See Fig. 1). Clinically, this situation is encountered only occasionally, for example in cases with a defect of the incus after operative removal in a previous simple mastoidectomy, also in cases with atresia of the external auditory meatus and middle ear malformations. Stapes fixation does not only cancel the effect of the tympanic membrane and of the ossicular chain, but also causes a certain interference with the hy-

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di[illegible]dynamics of the inner ear which will manifest itself in the Carhart notch (see Fig. 2). The total defect of the tympanic membrane eliminates to a large extent the sound pressure difference between the two windows (see Fig. 3); *furthermore it produces sound reflection on the bony wall of the* promontory instead of the sound absorption which under *normal circumstances occurs through the intact tympanic* membrane for the main speech frequencies in a very high percentage (see Fig. 4).

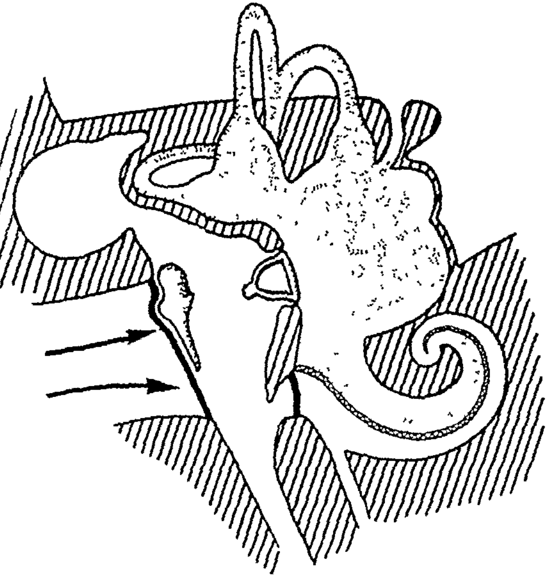


Fig 1

Chronic otitis media causes pathological and functional defects of various degrees, sometimes of minor extent only. Adhesions frequently lead to sound protection of the round window by adhesions, or to a partial sound conduction directly to the stapes, as Zöllner has shown (see Figs. 5, 6). I must, however, emphasize that often pathological processes will improve the hearing ability, for example secretions and granulations, which close a perforation or have the effect of an artificial drum membrane, or especially masses of cholesteatoma which in a defective ossicular chain produce a certain amount of sound conduction to the oval window. The indication for tympanoplasty, therefore, is often given even with relatively good hearing and with a small conductive component only, in order to cure a pathological process or to prevent a

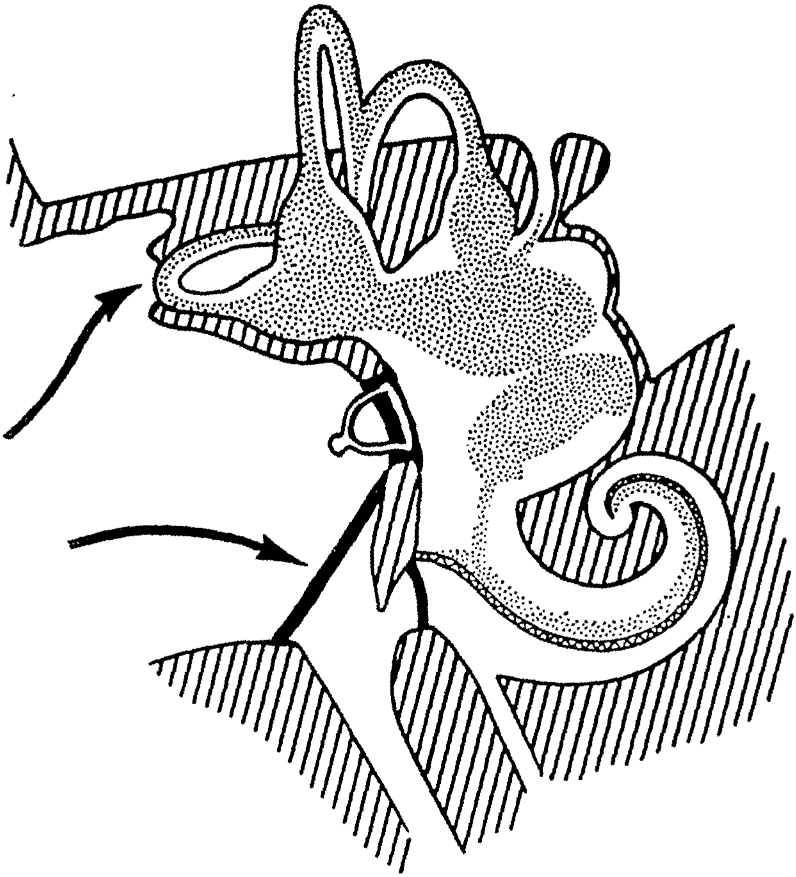


Fig. 2

reinfection of a dry middle ear. Today we are interested in a definitive cure of all cases of chronic otitis media, especially those with good middle ear function so that further destruction of the middle ear, or damage to the function of the inner ear, can be prevented. It is self evident that the removal of the pathological changes is the first task of a tympanoplasty; thereby it might become necessary to lower the functional performance of the middle ear to the extent to which it is based on the pathological processes, before it can be rebuilt again.

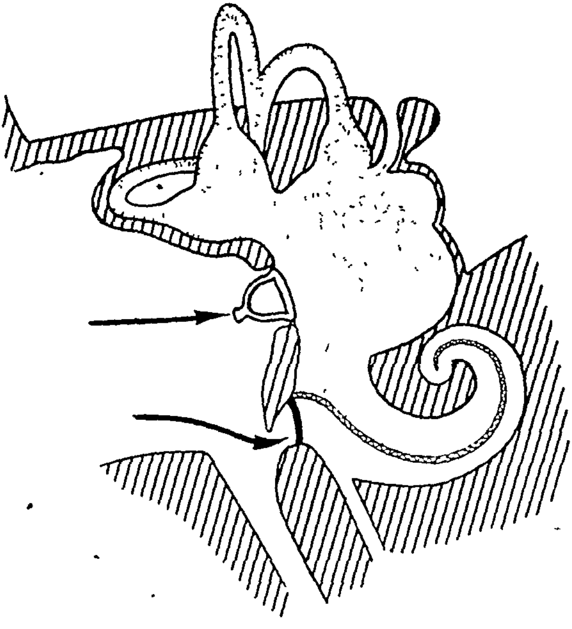


Fig 3

Theoretically the possibility exists, that disturbances in the transmission of the acoustic stimuli through the middle ear to the organ of Corti might not be recognized. First Wever and Lawrence, later on Ranke, Keidel and Weschke, Gisselson and Richter, have shown in animal experiments that the complete blockage of both labyrinthine windows leads to a very pronounced cancellation of the hydrodynamics in the cochlea, to a very marked reduction of the cochlear microphonics. Clinically, this situation must lead to the assumption of a marked damage to the inner ear. From the experiments of Ranke, Keidel and Weschke we have, however, also learned that in order to bring about this effect the round window niche must be completely closed right up to the round window membrane without any interposed air bubble. If a large cushion of air is left in the round window niche the hydrodynamics of the round window will not be interfered with. It is difficult to say whether in clinical practice interference with the hydrodynamics sometimes is actually mistaken for inner ear damage. I do not, however, believe that the question is of great practical importance. From the operative findings in more than 500 tympanoplasties I can state that a complete closure of the round window has only once been observed and that it, therefore, will be still rarer in combination with complete or incomplete closure of the oval window.

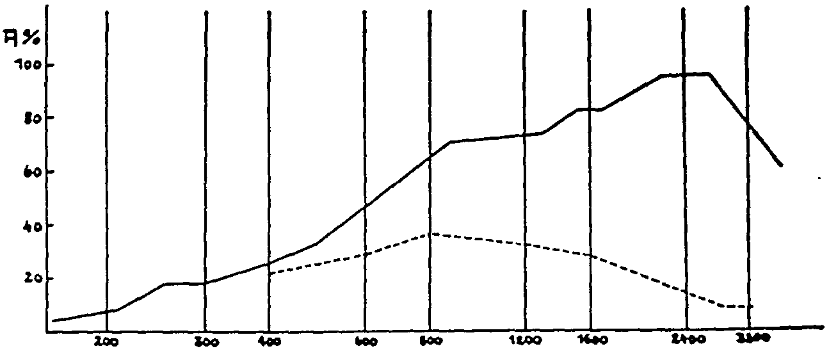


Fig. 4. Sound-absorption of the tympanic membrane ———— normal; ………… complete defect of the tympanic membrane (after Menzel).

In contra-distinction to otosclerosis, where we have almost exclusively to deal with ankylosis of the stapedial foot-plate, chronic inflammatory and adhesive changes of the middle ear cause the most heterogeneous functional findings; therefore, the tympanoplasty calls for constant variations in the operative technique. The audiological findings, however, in these operations frequently lead to certain operative ways of reconstruction of the sound conducting apparatus of the middle ears which I have, therefore, classified into five types. In principle each tympanoplasty has to be carried out according to one of these types (see Fig. 7).

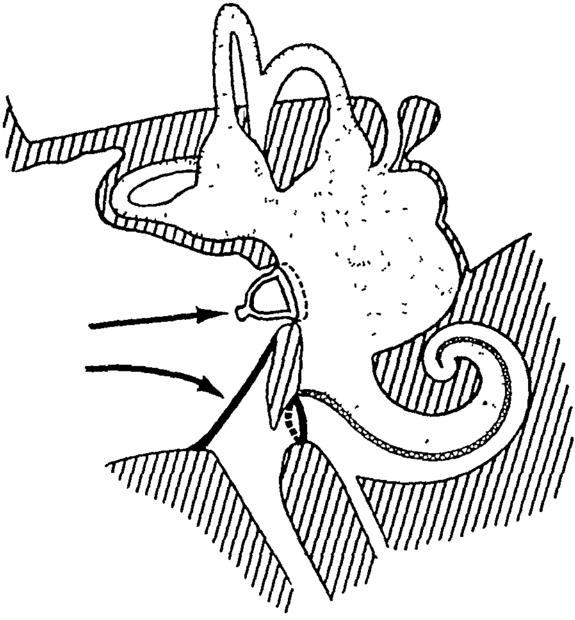


Fig 5

Type I. Practically rebuilds a normal middle ear; a tympanic membrane with both functions, sound-protection for the round window and the scala tympani, and sound pressure transformation over the ossicular chain to the scala vestibuli.

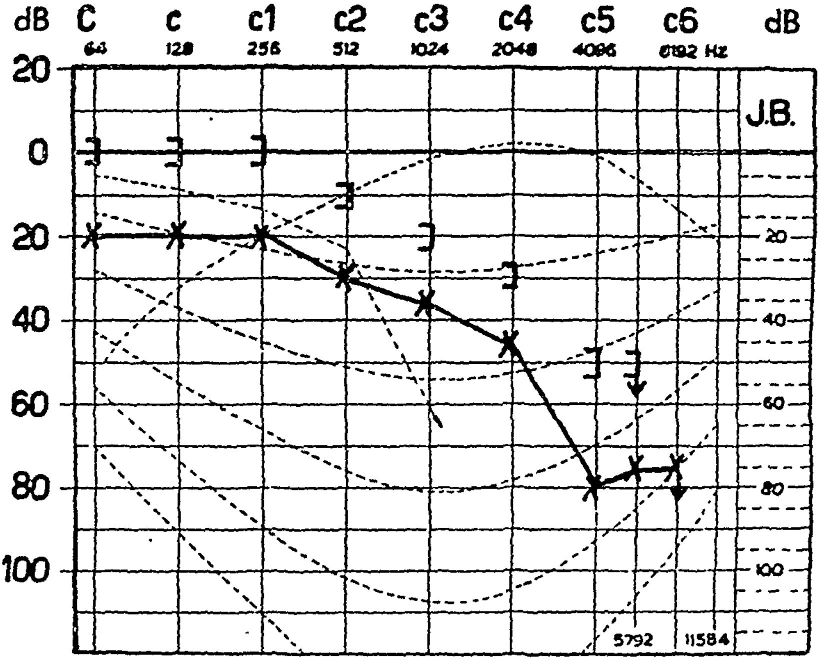


Fig. 6. Spontaneous columella-effect after frequent otitis media during infancy. Tympanic membrane transparent, mobile, on stapes adherent. Defect of the long process of the incus. Presbyakusis.

Type II. Tries to preserve, in spite of slight defects of the ossicula, a middle ear of about the normal size and depth between tympanic membrane and promontory, including the epitympanum.

Type III. In this type large defects of the malleus and incus make the removal of the ossicular chain and of the epitympanum necessary. The tympanic membrane, in order to function as sound pressure transformer, must be directly connected with the head of the stapes. The frequent spontaneous connection between tympanic membrane and the stapes through adhesions has been named "Columella-effect" by

Zollner, "Myringostapediopescie" later by Juers. In 1901 Matte operatively achieved and published such a connection. Through a reposition of the tympanic membrane over the stapes and the lateral semicircular canal, the mesotympanum is narrowed in the promontorial region, and in this way there is an increased danger of formation of adhesions.

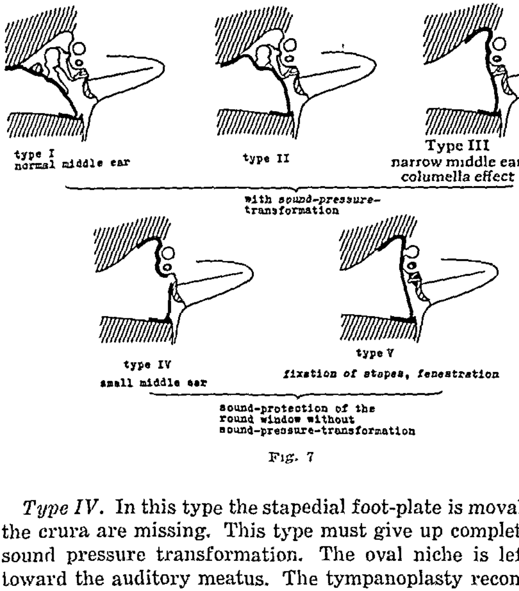

type I
normal middle ear
type II
Type III
narrow middle ear,
columella effect
with sound-pressure-transformation
type IV
small middle ear
type V
fixation of stapes, fenestration
sound-protection of the round window without sound-pressure-transformation

Fig. 7

Type IV. In this type the stapedial foot-plate is movable, but the crura are missing. This type must give up completely the sound pressure transformation. The oval niche is left open toward the auditory meatus. The tympanoplasty reconstructs a middle ear consisting only of the tube and the hypotympanum with sound protection for the round window. This operation was first done by Moritz.

Type V. In this type the stapedial foot-plate is fixed and a fenestra novovalis is necessary.

In theory we can predict quite accurately the results of these tympanoplasties. In Types I-V the tympanic membrane fulfills both functions, sound protection and sound pressure transformation. If this is done in an ideal manner, no conductive loss should remain; it should be reduced to zero.

The effect of the ossicula in the transformation of the sound pressure is very small, after von Békésy it is only 1:1.3. The *effect of the tympanic membrane upon the oval window is,* however, 1:17, corresponding with the relationship of the sizes of the two surfaces to each other which had already been measured by Bezold, Edelman and also by Frank. We have converted these relationships into decibel figures, Güttner.

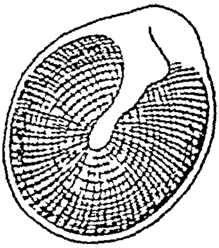
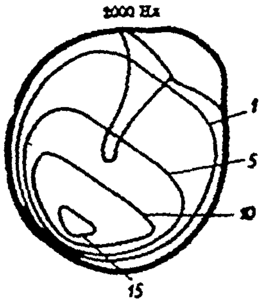
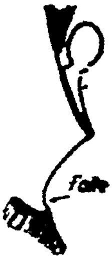


Fig. 8. Network of collagenous fibres.



Curves of equivalent amplitudes of the tympanic membrane (frequencies below 2 400 Hz) (after G. v. Bekesy).



Fold of the maximum of amplitude (after G.v. Bekesy).

The loss of the leverage effect of the ossicles, which occurs in Type II, and especially in Type III, amounts theoretically to 2.5 db only.

If the loss of the ossicles amounting to 1:1.3 is combined with that of the tympanic membrane amounting to 1:17, then this product amounts to 1:22, which is converted into decibel figures of 27.5 db. The loss of the entire sound pressure transformer, therefore, causes an air conduction loss of 27.5 db. This theoretical figure closely corresponds with the figures obtained from thousands of fenestrations in which one must sacrifice the sound pressure transformer for the scala vestibuli, and can only preserve the sound protection for the round window through the tympanic membrane; therefore, the typical result of a fenestration operation lies between the 25 and

30 db hearing loss for air conduction, as compared with bone conduction (see Davis and Walsh). Types IV and V, which are audiologically identical with a fenestration for otosclerosis must theoretically reach these figures.

Whether these results will actually be obtained depends to a large extent upon the quality of the new tympanic membrane. The tympanic membrane is a stiff membrane which vibrates around its lower fold, as has been shown by von Békésy. The stiffness of the membranes is due to the contained collagenous fibres which, as Kobrak pointed out, cannot be stretched (see Fig. 8). If large parts of the pars tensa are well preserved in quality, the effect of the sound pressure transformation and also of the sound protection can be restored more easily. At the place of the perforation or atrophy we cannot restore the natural structure of the tympanic membrane; therefore, we must be aware of a deterioration in the quality of the new tympanic membrane as compared with that of a healthy one. If, for instance, the effect of the sound

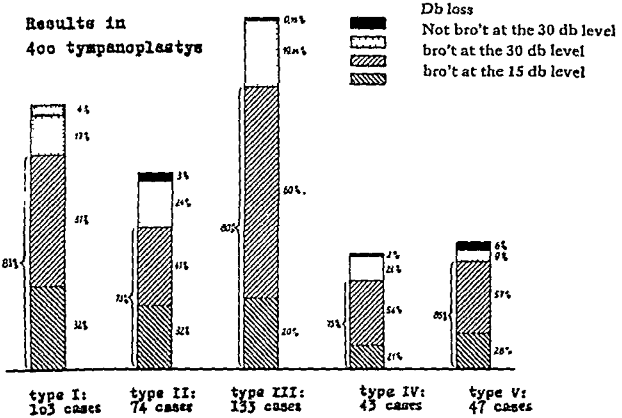


Fig. 9

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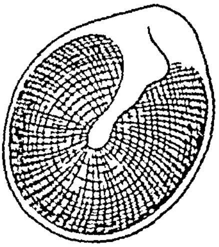

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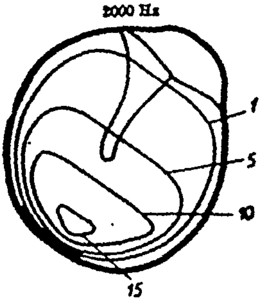

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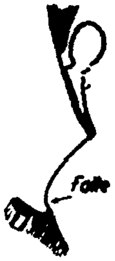

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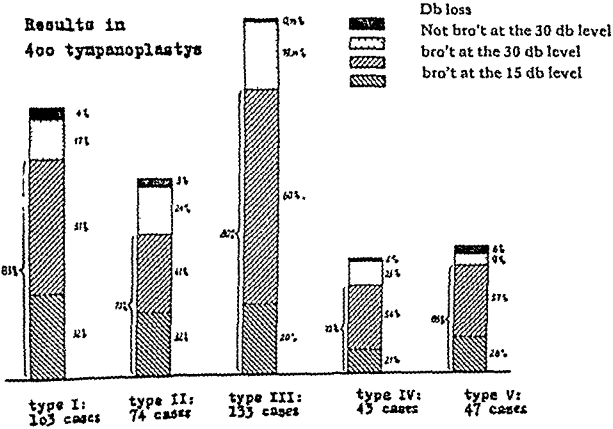

Fig. 9

pressure transformation of the drum membrane is reduced from 1:17 to 1:5, we get a hearing loss of 13 db. If at the same time the leverage effect of the ossicular chain amounting to 2.5 db is lost, we get altogether a conductive loss of about 15 db. The result in a tympanoplasty of Types I-III, as in stapes mobilization, with reconstruction of the sound pressure transformation is, therefore, called good as long as the conductive loss is below 15 db for the frequencies of 500 to 2000. If there is a conductive loss from 15 to 30 db for the Types I to III, I call the result only satisfactory.

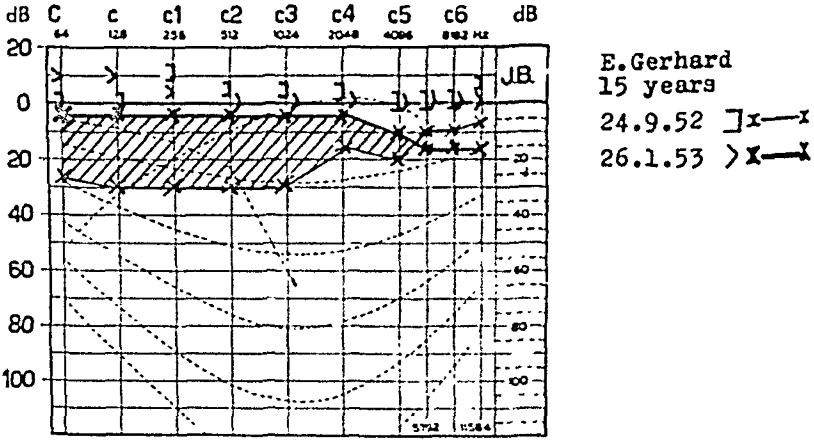

Fig. 10. Small central perforation. 30.9.51 Tympanoplasty Type 1; Ossicular chain intact. Free skin-graft, overlapping. 30.1.53 New tympanic membrane mobile.

If Types IV to V, as in fenestration, there are no such differences. Since in these types only sound protection is present, all the results can be called good which show a conductive loss of less than 30 db. All results of an audiological operation on which the conductive loss is greater than 30 db for the main area of speech, is theoretically to be regarded as unsatisfactory, even though the operation produced a definite increase in the hearing. I believe that I gave you a theoretically well founded classification of our audiological operations.

In demonstrating the results of 400 tympanoplasties, I ask that you take into consideration the fact that therein are shown all operations, even those from the early stages of my operative endeavors. In order to study the limits of the indications and the methods of the operative technique, I have operated on all cases of chronic otitis, without exception, providing there were no contra-indication through the danger of endocranial complications. Even ears with marked and fetid suppuration, heavy granulations and extensive cholesteatoma were operated upon. Almost invariably a one-stage operation was performed; only fenestrations in Type IV were done in a two-stage procedure (see Fig. 9).

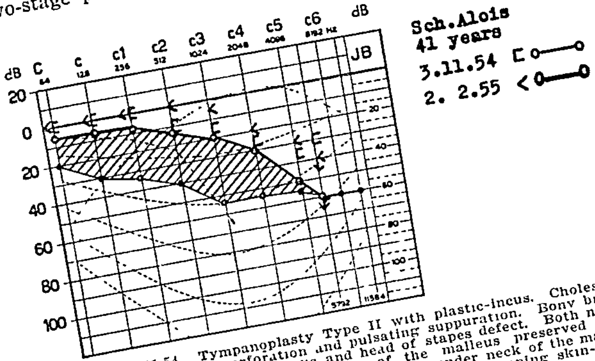

Fig 11. 16.11.54. Tympanoplasty Type II with plastic-incus. Cholesteatoma large marginal perforation and pulsating suppuration. Bony bridge removed Head of malleus, incus and head of stapes defect. Both niches dissected from granulations. Handle of the malleus preserved with tendon of tensor tympani muscle Palavit-staff under neck of the malleus to neck of the stapes Implantation of amnion Overlapping skin-graft 2 2 55 New tympanic membrane mobile.

It can be seen that a high percentage of these patients have reached the 30 db line, theoretically for the Types I to III a satisfactory hearing ability; for the Types IV and V a good hearing ability. In the Types IV and V we even find a considerable number of patients in whom, without the apparatus of sound pressure transformation, results have been achieved, which in Types I to III would also have been called good. Since, for instance, in Type IV, the stapedial foot-plate lies open toward the auditory meatus and has not been touched, the good result can be due only to an increase in the sound protection for the round window. In a small middle ear, built from the tubal corner, hypotympanum and round window niche, it is evidently quite often possible to increase the sound protection as compared with that provided by the normal drum if a dense scar is formed in the transplant which closes the reconstructed cavity. Still further increase in sound pressure difference between scala vestibuli and scala tympany may be obtained by sound reflection in the cavity to the fenestra ovalis or nor-ovalis. If in comparison to the "good" result in the last two types, the number of the "good" results in Types I to III is relatively small, we must take into consideration that in many instances, especially in Type I, the air conduction curve, after healing, almost reaches the bone conduction curve, and only a conductive component of about 5 db will be left, which does not happen in Types IV and V. We must also consider that although the possibility of restoration of the sound pressure transformation exists in most of the tympanoplasties, defects of the ossicula are always signs of a long lasting granulating disease of the mucosa on the promontory and on the inner surface of the tympanic membrane. In this way the chances for the highest functional quality of the new middle ear are reduced.

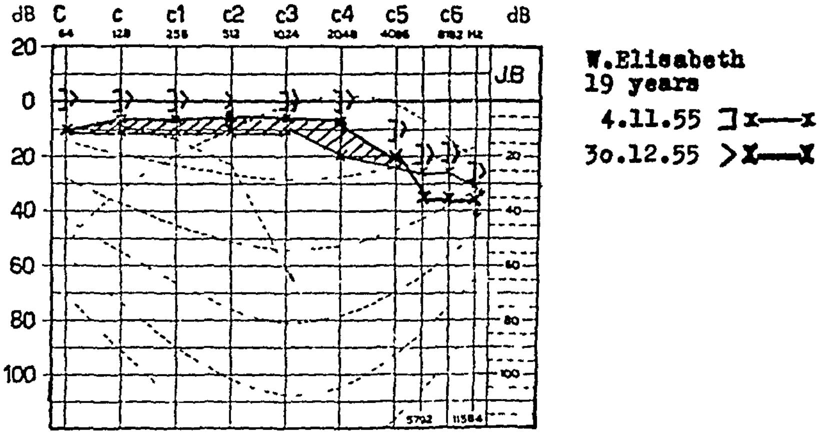

Fig 12 10 11 55 Tympanoplasty Type III Defect of the membrana flaccida, pars tensa preserved, cholesteatoma in the attic, matrix enclosing the ossicles Bony bridge, incus and head of the malleus removed Free skin-graft

In what manner do we have to plan and carry out the operation if we want to do it according to these principles? It is the first law in a tympanoplasty, like all other audiological operations, to remove no element of the structure of the middle ear before the whole middle ear has been examined with the operating microscope and the general plan of the operation, namely the type of the tympanoplasty, has been established. Frequently it is impossible to determine the type of operation before one has actually started. Remnants of the tympanic membrane, adhesions and granulations prevent us from reviewing the whole mesotympanum through the auditory meatus, and also prevent us from determining the acoustic functional ability of the two windows. The opening of the tube is often closed and the patency of the tube, therefore, not demonstrable. The severity of the infection of the mucous membrane might show considerable variations; for instance, there might be heavily granulating mucosa in the hypo and mesotympanum, whereas the antral mucosa might be almost without irritation or vice versa. For the microscopic examination of the tympanic and retrotympanic cavities without even the most minimal interference with the functional struc-

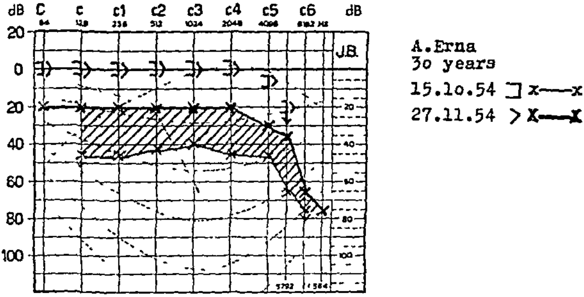

Fig 13 2-11 54 Tympanoplasty Type IV Large cholesteatoma, total perforation, fetid suppuration, defect of the head of malleus of the incus and of the crura stapedis footplate covered with epidermis Large fistula on the lateral semi-circular canal Round niche dissected out of granulations Free skin-graft 27 11 65 New tympanic membrane mobile tures of the middle ear I have established three control entrances:

1. Control of the *antrum*.
2. The upper control of the middle ear.
3. The lower control of the middle ear.

For the control of the antrum I enlarge the bony auditory meatus a little bit backward and then open the antrum through a drill hole only closely behind a broad bridge which gives protection to the whole epitympanum and the ossicles. From here the pathological process in the antrum and also in the aditus ad antrum can be visualized.

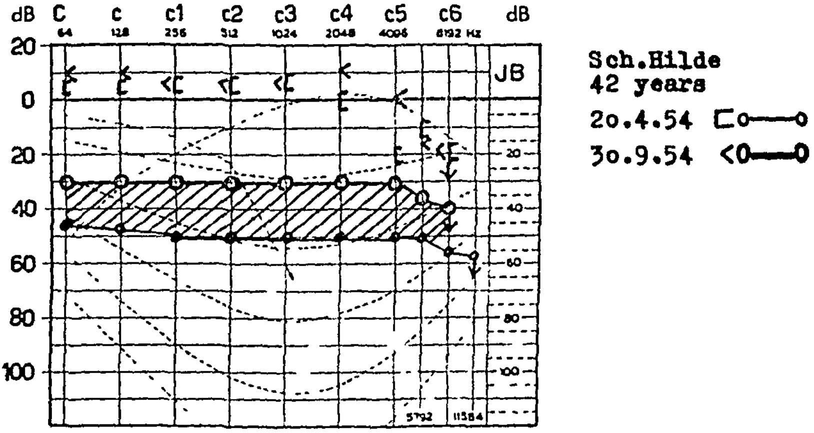

Fig 11 Otosclerosis and central perforations of the tympanic membrane 27 1 51 Tympanoplasty Type V. Small cholesteatoma in the attic, bony bridge preserved, fenestra in the semi-circular canal prepared Free skin-graft 17 9 51 Fenestration Brother with success fenestrated for otosclerosis only

The upper control of the middle ear is done through the removal of Shrapnel's membrane, which seals off the epitympanum but does not participate in the transformation of the sound pressure. From here control of the incus and malleus can be accomplished, especially the control of the long process of the incus, which is easily destroyed by granulations from the oval window niche and from the epitympanum. The operation can, therefore, be shortened if one examines at an early stage this weakest point on the ossicular chain. The stapes and oval window niche can also be reached from here. After removal of a small marginal portion of the bridge with a diamond burr, one can satisfactorily visualize the epitympanum and a small sac-like cholesteatoma in Prussak's space can be easily enucleated.

In a larger cholesteatoma where the matrix is not yet attached to the whole attic and does not cover the ossicula but still can be removed as a sac *in toto,* I cut through the bridge with the finishing burr either below the tegmen tympani or closely above the facial canal, depending upon the place where the cholesteatoma sac extends into the depth. In this manner the ossicula can be preserved under the protection of a large part of the bridge.

The lower control of the middle ear is known through my method of fenestration, because I never create a new window before I have microscopically controlled, with preservation of the whole middle ear, the tympanic cavity; and especially both labyrinthine windows. Close to the floor of the auditory meatus, between annulus osseus and the lateral semicircular canal, more precisely, between chorda tympani and nervus facialis below the tip of the short process of the incus, an opening into the sinus tympanic is established with the diamond burr which will lead in a typical manner directly to the stapedial tendon. After the enlargement of the opening, the oval niche and the hypotympanum to the round niche will come into view. On still further enlargement toward the floor of the auditory meatus even the membrane of the round window can be identified on a view from behind and below.

Entrance to the oval niche can be gained from this point to such an extent that with this approach the direct mobilization of the stapedial foot-plate, especially around the base of the anterior crus, can be carried out. In this manner we are able to prove the function of both windows, before the incus is removed and possibly the crura of the stapes are infractured. It is furthermore possible to smooth out, with a fine diamond burr, a round window niche which had been blocked with connective tissue. Thereafter an implant serving as substitute for the mucosa can be laid directly over the round window membrane.

In large perforations of the drum with both window niches exposed to the direct view, not all these controls are, of course, necessary. If, however, the posterior part of the drum over both windows is preserved and contains a good layer of collagenous fiber, it is in my opinion not advisable to lift it out of the annulus. Through the perforation it has already lost some of its stiffness, and in this way it might suffer a further loss of its functional quality.

I think that these operative controls are even then necessary when the chronic otitis shows findings which are most favorable for a tympanoplasty such as: central perforation; pars tensa well preserved; no discharge; mucous membrane of the middle ear, as far as visible, not irritated; good increase in the hearing ability even in the threshold audiogram by closure of the perforation through an artificial drum. The marked increase in hearing with an artificial drum is often not successful at the first attempt; but if we are successful at least once, it is evident that both labyrinthine windows are able to function properly. The lack of an increase in hearing after repeated attempts does not, however, prove that there is a fixation of one of the two windows; there could be a blockage of one of the window niches, or it could have different other causes. Even under the most favorable conditions of a central perforation one often finds on control of the sinus tympani, the round window niche or the epitympanum loose granulations which completely fill up these narrow spaces. I have observed young patients for many years, before the operation, who had first a very good hearing gain on closure of a perforation which later on decreased. On operation a progressive scarring of such granulations could be seen. Through an operation which solely closes the perforation (myringoplasty) one can easily get a good initial result; but there is great danger that after several years, with progressive scarring, the hearing ability will go down below the preoperative level. A revision of the first operation is less likely to succeed, because extensive masses of scar tissue must be removed and large areas of the bony wall will be without a mucosal lining. Myringoplasty with Thiersch grafts were tried 70 years ago by Berthold and Tangemann, but were abandoned.

The results in the different types of cases indicate that the theoretical considerations regarding the audiology of these operations are correct. We shall now see a sample of each of these different forms (see Figs. 11, 12, 13, 14).

The theory and practice of these operations which show a great variation I could only briefly demonstrate. They can only be done under the microscope and they are very strenuous and time consuming; but all this is worthwhile, because a definite healing of the chronic otitis media and a large percentage of good functional results can be achieved. I believe that all audiological operations are interrelated: fenestration, mobilization and tympanoplasty in chronic otitis media, and in adhesive changes after middle ear infection; malformations of the middle ear and atresia of the external auditory meatus belong to the same group of cases.. On this occasion the operation for the middle ear follows the same principles as the tympanoplasty; with a movable stapes a columella-effect might be advisable and with ankylosed stapes, a fenestration.

SIXTH INTERNATIONAL CONGRESS ON OTOLARYNGOLOGY.

The deadline for submitting titles of papers and films for presentation at the Sixth International Congress of Otolaryngology is Oct. 1, 1956. Requests for places on the scientific program must be received before that date together with an 800-word abstract of the paper. Applications and abstracts should be sent air mail to the General Secretary, 700 N. Michigan Ave., Chicago 11, Ill., U. S. A.

"SICK PEOPLE IN A TROUBLED WORLD."

HOWARD A. RUSK, M.D.,*† (By Invitation)

New York, N. Y.

I would like to first discuss briefly rehabilitation in general and then talk about some of our mutual interests. Rehabilitation has really become a $64,000 word. It means so many things today that it means very little. It may be used in discussing a program for the economic recovery of a country, a business; it may mean the bringing back to optimum of a severely disabled individual. In our own department we call rehabilitation the third phase of medical care, the first phase obviously being prevention; the second definitive medical and surgical care, and the third phase is that which happens to the individual after the stitches are out and the fever is down.

I would like to outline for you briefly the problem that you and all of us in the health services have had a great part in creating. Along with improved knowledge of nutrition and a better standard of living, we have raised the age expectancy in this country from 46 at the turn of the century to, as of last month, the Biblical three score and ten for the first time in the history of the world. This has created a problem; it means that today in the United States we have 14,250,000 beyond the age of 65. By 1980 with our present medical knowledge alone they will exceed 20,000,000. Of this 14,000,000 or more, one-third have no income at all. One-third have less than $500 a year, and of the remaining one-third, 50 per cent are self-employed. A man today of 65 has a life expectancy of just over 15 years; a woman 17.6 years. This has created a special problem best summarized in a sentence by George Morris Piersol some years ago, when he said, "We have added years to life; it is also our responsibility to add life to years." The first problem, therefore, is that of retirement.

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* Presented at the Sixtieth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Montreal, Canada, May 15, 1956

† A verbatim transcript of an extemporaneous address

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Everyone knows that age is physiological and not chronological, that some are old at 50 and others are young at 80. By the willy-nilly employment of the retirement practice that is currently in vogue, I feel that we are wasting the most precious human resource that we have. That resource is wisdom! People are not born wise; you may be born brilliant, but wisdom comes only with experience and experience comes only with time.

I wonder how world history would have been written had this retirement policy been adhered to. It would have meant that Winston Churchill would have retired a year before the Battle of Britain; that Mr. Baruch would have retired 21 years ago, before the rubber program and before the atomic energy program, and we would have lost all of those years of his wise counsel.

I, for one, feel that because we in medicine have taken the leadership in prolonging life, we also must provide leadership in meeting this problem; and I think that any country which can solve the mysteries of electronics, radar and atomic fission can certainly put the pieces together to know who should continue to work without detriment to his own health or danger to his fellows. Industry is certainly able to know who can continue to work and be valuable as a producer. This is the No. 1 problem that we face.

Second, we have all of the problems of disabilities that come with an aging population. We became tremendously disturbed in this country about the problems of infectious disease and have done something about it.

In an epidemic of polio we have, I think, approximately 52,000 cases. Today in America, not including Canada, we have 1,500,000 individuals who have had strokes of apoplexy. In the past they were felt to be hopeless; they are not hopeless at all. Winston Churchill again is a good example.

We just finished an anlysis of our first 1000 at Bellevue. The average age was 63. The average time from stroke to beginning training, nine months. The average training time seven weeks; 900 of the 1,000 we were able to train to the point where they could meet all the needs of daily living and ambulate and live non-institutional lives, and 400 went back to some kind of gainful work.

In this third phase of medicine every time we make medical advancement we compound the felony, as far as we in rehabilitation are concerned, because people stay older longer to get all the disabilities that come with age. I think, too, that this realization brings a new and very basic concept which, I believe, that we in medicine have failed to remember, just as the general population has failed to remember. Not for hundreds of years has society paid for strength. It pays only for two things: the skill in your hands and what you have in your head; but if you are honest and let your subconscious reflection prevail, physical perfection and ability will seem synonymous.

The average person uses only 25 per cent of his physical capacity in daily living, but the reason we can do a pretty good job in the practice of medicine is because nature has given mankind such terrific powers of healing and of physical compensation. The blind man sees with his sense of touch and with his sensitized hearing. Put that man in a job where he can use those overdeveloped senses, and every experiment has shown that he will outproduce the normal man from 20 to 30 per cent. Put the paraplegics at a bench job where they can use the hypertrophied muscles in the upper arms and shoulders, and they will kill the ordinary worker doing the same job and outproduce him every day in the week, because they are working with hypertrophied muscles.

I will give you an illustration of just how easy it is to forget. About four years ago I was at a meeting in a western city and had a call from a young woman physician. She asked whether she could see me. "Certainly! Would you like to come by the hotel?"

"I am a paraplegic in the hospital," she said. "It is impossible, but you are speaking at the hospital tonight. Can I see you after the meeting?"

I said, "Certainly."

"You will recognize me. I am a paraplegic. I am in a wheel hair."

Her story was this: She was 32 years old, had five years aining in medicine, neurology, psychiatry at Bellevue, had me back to her home city, was instructor in medical school, nd was assistant to the leading psychiatrist in this city. ourteen months previously a psychotic policeman had come nto her office looking for the boss. Not finding him there he ad pulled out a gun and shot this girl five times. One bullet ransected the cord at T-10. The intestines were punctured n dozens of places; also a bullet wound through the liver and ne in the right thorax. She had magnificent treatment; otherwise she would not have survived. The internist took care of he antibiotics, the blood, the electrolytes and the fluid; and he general surgeon sewed up the rents in her intestinal tract

With advice of work simplification and home management, many women, espite severe disabilities, can assume their roles as homemakers and stopped the bleeding in the liver. The neurosurgeon did a laminectomy. The surgeon stopped the bleeding in the chest. This was 14 months ago, and this was the first night she had worn a dress.

I said, "What is your treatment?"

"Massage to the lower extremities 20 minutes three times a week."

I said, "Are you practicing medicine?"

She looked at me like I was crazy. "My surgeon said to me, 'let's face it; you are all washed up as far as medicine is concerned.' One day one of my colleagues came by and said, 'Why don't you come down to the clinic on my day? You are perfectly capable of filling out X-ray requisitions.' "

I said, "I've learned something. I always thought you practiced medicine with a trained mind, dedicated heart, eyes, ears, your hands, personality and your voice. All of those things you have. I didn't know that feet were a necessity."

She smiled wanly and said, "I don't know what I am going to do. I have a $70 month pension, a two-year-old baby; my husband has left me and I owe $3,000 for a hospital bill." I said, "Certainly something can and will be done."

I went home the next day and worried about this girl all weekend, and Monday afternoon called her and asked her when she was coming to New York to take her new job.

She said, "What job?"

"You have just been given a two-year appointment as assistant resident on the rehabilitation service."

She said, "When do you want me? Mind you, I have never been out of the hospital."

I said, "When convenient."

In 48 hours she arrived. We did not put her in the hospital. We put her in the residents' quarters, and placed her in the training program, training herself half a day and seeing patients half a day. That was the 25th of March, 1951.

On Labor Day she walked on her braces and crutches to a taxi, went to the airport, walked up the ramp, flew back, and spent two weeks with her child. She finished training, qualified for the Boards, took her examination and finished in the upper 10 per cent, and that young woman now is the Acting Chief of the Rehabilitation Service on the Neurological Service in the National Institutes of Health. At home in a great medical center, one of her own faculty forgot what was needed in the practice of medicine. Now that girl is a better doctor in this field than any whole person can ever be, because she will teach both by precept and by example.

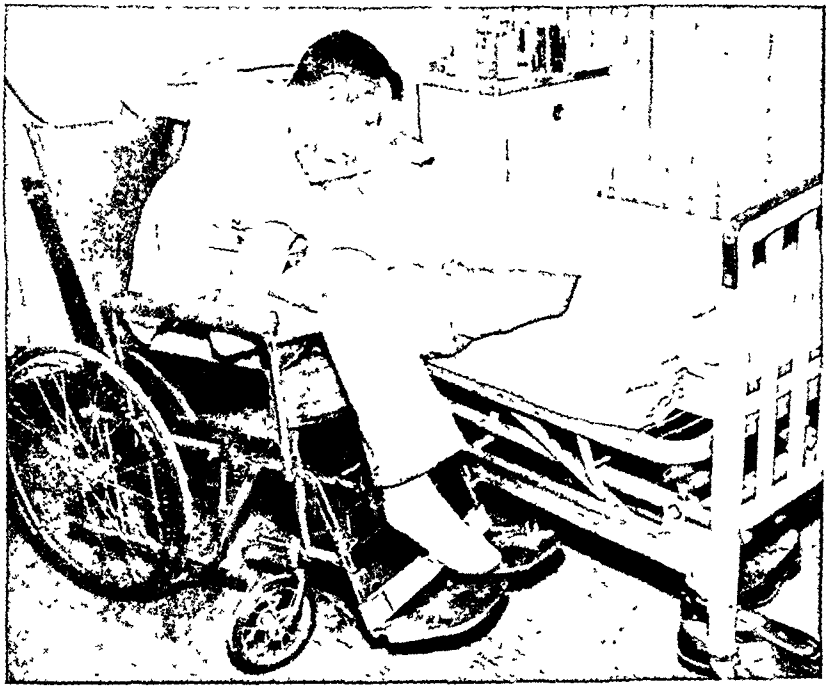
What is this rehabilitation service? What do you do? It is very simple. Our concept is that we are a service department like the laboratory and the X-ray, and in the definitive stage of medical care, we can act in a consultant capacity.

At Bellevue, for example, we see all elective amputations before the member is amputated. We tell the individual why he cannot keep the stump on the pillow, and the exercise he needs to prevent the flexion contractures. We tell him what an artificial limb is, how much it costs and how long it takes to get it; that he has to be trained to use it, and often we will bring an amputee with us to demonstrate. In the older age group, with poor balance and a problem in the other leg, or a cardiac problem, we will begin to discourage him on a prosthesis even before he is operated upon, and tell him what he can do in a wheel chair.

When training becomes primary and definitive care secondary, then we like to accept primary responsibility. Patients come to us; we are a professional service and not a technical service. We reserve the right, as you do, to examine the patients referred to you and either agree or disagree with the diagnosis, and that we do.

To show you the need of this particular phase I will cite briefly a case that came to us two years ago. A young man who had been in a terrific automobile accident with severe brain injury, unconscious nine months, tube fed, three craniotomies, contusions of the brain, and when he finally woke up he had a severe cerebral ataxia and a terrific speech defect.

You know the brain syndrome and how difficult and discouraging it is to treat, probably one of the most difficult that we see in adults. He finally got on his feet. He was sent as an ambulatory patient to a training center in the city in which he lived; 12-pound leads were put on each shoe and wrist leads to try to decrease the ataxic movements. He went into speech therapy, but his mother complained bitterly that this boy wasn't getting along in speech, and the reason he could not speak was that he was dyspneic, and something had to be done. They were told over and over again that this was a severe injury, that it took a long time and that they must not be impatient.


Such simple things as getting from the wheel-chair to the bed are the foundations of self-sufficiency for the severely disabled.

When the Fellow on our rehabilitation service did the original examination, he was interested to note that this boy had a dextrocardia. He was also interested to note there were more intestinal than breath sounds in the left thoracic area, and a barium swallow showed about two-thirds of the intestinal tract in the left pleural cavity. Walking with 12-pound leads is not specific for diaphragmatic hernia.

He was sent back to the surgeon, who closed the rent, and strangely enough the boy could speak a great deal better. This is the first phase of treatment; then we do a careful muscle study; range of motion and definitive muscle examination, but the heart of our program is simple. We test for 137 inherent needs in daily living, things that you do every day subconsciously. The following questions are asked: Can you brush your teeth; can you comb your hair; can you feed yourself; can you dress yourself; can you turn from side to side in bed; can you put on your own braces; can you get from bed to wheel chair; from wheel chair to toilet; from wheel chair to bath tub; from bath tub to wheel chair? If you walk, can you step up and down the curb? (If you cannot, walking does you very little good.)

About five years ago, Dr. George G. Deaver, my associate at New York University, reviewed 500 cases of polio, 16 years or older, which had the disease five years or longer and were confined to their homes, to ascertain why. He found 70 that with adequate training would not need to be thus confined.

The first girl we brought in was 28, had polio when two. She was a quadriparetic, she had had 26 assorted surgical procedures, six spinal fusions, could walk only with a crab-like gait on crutches, and could not get up and down a curb. She had had a home teacher, learned to type, and could type 30 words a minute accurately. This did not help her, as she could not go out, nor could she get up and down the curb to cross the street. It took seven months to teach her a technique by which she could get up a curb by swinging the leg back, catching the heel and turning a certain way. I happened to be in the gym the day she made it the first time. The patient screamed like a cheering section of the football game. Once she learned the trick she did it a dozen times the first day, 25 the second and 50 the third. That was Friday; she went home on Monday and took a full time job at the switchboard at

Bellevue; that was five years ago, and she has never been late a morning; never missed a day. She waited 26 years, because there was an eight-inch wall around her house.

I tell the story because it exemplifies the necessity for attention to detail and this is the principal factor in the training program for rehabilitation.

Wherever possible group instruction and training is given at the Institute of Physical Medicine and Rehabilitation.

Then our patients are psychologically screened, and have an interview with our vocational counsellor. We begin to talk about work as soon as we see the patient, and then the patient comes before the whole staff. The program is planned, and these individuals go back to school under a planned program, going to class five hours a day.

Hemiplegia is the most common disability we see. The average training time of hemiplegia is six weeks. The most difficult problem in training the hemiplegic which you see is aphasia. About 40 per cent of the patients we see are aphasic. We have learned a number of simple things about the problem of aphasia and not too much about the long tedious training.

In the first place in this country we have fewer speech therapists than any of the other co-professional personnel. I should say that if every school that has a class of speech therapists doubled its class every year for the next ten years there would still be too few speech therapists.

The first thing that must be done for the aphasic is that he must be assured that he can be helped. We have seen patients who have been in the most absolute agony for two to four months because of aphasia. Nobody had taken the time to explain this simple thing.

It takes careful evaluation by a trained person to know the type. Unfortunately, most of those we see are mixed, and it takes continuous training over a considerable period of time. Let me suggest one thing in the treatment of aphasia: Do not be too positive in your prognosis, because when the edema subsides in the healing area about eight weeks after the original cerebral insult, the outlook and prognosis often change totally. The same is true for the diagnosis of organic brain damage in the hemiplegic patient, because until this first acute phase is over, you cannot tell how much the patient will be able to do.

Paraplegics have been a very interesting group with us, and we have now seen nearly a thousand. The program started in the services during the war. In World War I we had 400 paraplegics; they were no problem then, because one-third never got back to this country. Among those who did return another third did not survive 60 days. The mortality was more than 90 per cent in the first year. There are only two living today from World War I that I have been able to find, and they were not total, but partial.

In this last war from combat we had 2500. They were a real problem. This time knew about urology and nutrition and the management of decubitus ulcers and we had antibiotics. They did not die. They were young men who had given the

It is difficult if not impossible for the non disabled to understand the emotional satisfaction which comes to the paraplegia the first time he stands again

best of themselves to the country. They had strong arms and good minds. They wanted to live the best lives possible with what they had left.

I can say that the dynamic program in the VA hospitals is magnificent.

Out of the original 2500, 1763 today are living in their own homes, driving their own cars and 80 per cent of that group are either at school or in some kind of job.

It might interest you to know, however, that the case load in VA hospitals has not decreased by a single case. For every paraplegic who has gotten out, another from the casualties of civilian life has taken his place. This is a hard lesson to learn.

In the early days after the war we all thought about disability in terms of the veterans. The veterans are a drop in the bucket. The first ten days on the Normandy beachheads we lost 11,000 men killed and wounded. In the same ten days we had 26,000 civilians killed and disabled on the highways alone, and that was with gas rationing. The three days of the bloodiest fighting of the war were the three days at Iwo Jima. We have four times as many individuals killed or wounded by farm accidents alone each year in this country as were lost at Iwo Jima. I think the general figure now is ten to one.

The average training time for the clean paraplegic, T-10 or below, is 120 days. I say "clean" because it takes us twice as long to clear up the complications due to faulty early management, bed sores, kidney complications and contractions, all of which can be prevented, than it does to train the patient.

We have to change our thinking about the moving of these patients. We have seen nine patients in the last two years in whom we were perfectly sure that the cord was not transected by the accident but by the moving of the patient; because the textbook says that these patients should be moved in hyperextension. You do not know how these patients should be moved until you see the position of the fracture; therefore, they should be moved on a hard surface in a neutral position.

One of the most poignant stories I ever heard was one of a young Naval aviator who crashed the plane. He was able to crawl away while it burned. He had sensation and motion in his feet. He was picked up by the aid men who had read the book and who forced him into hyperextension. On the 15-minute ride to the hospital his legs went to sleep. When he arrived he was paralyzed; his cord had been transected en route.

The children's program is a particularly interesting one. Here, too, we have brought it on ourselves, because the children who formerly would have died, do not die today. We just finished an interesting series of spina bifida, and I think that every one who remembers the pre-antibiotic days, recalls an experience with this tragedy of the obstetrical ward. When the spina bifida child was born, you had to tell the parents this horrible thing, and the child was kept in the hospital. You and I knew that soon the child would turn, break the sac, get excoriations and ascending meningitis, and we would work vigorously together. We remember the days when you put urethral catheters in the spinal canal and washed it out with mercurochrome and anything we could lay our hands on. None of the spinal bifida cases ever recovered, and we felt relieved, and so did the family; but they no longer die. They live as paraplegics, and more than 80 per cent of them have good minds and strong hands and feel that they have a right to live the best life they can with what they have left.

We have just run our first series of 50, all types, and 35 are back in school. More than 80 per cent have bladder and bowel automaticity. Once braced and on their feet, they start to grow.

These are little children who will make their places in the world, because their life will depend upon the capabilities of their hands and their heads. Although they cannot run so fast, in some ways they have an advantage over normal children.

We have as consultants all of the specialties in medicine and one of the most valuable is the otolaryngologist. We could not make definitive diagnoses of speech disorders without you. Every child that comes on our service has a screening test for hearing acuity. We have a boy in the institute now who has spent eight years in an institution for the feebleminded, when he was only deaf. Once he was properly fitted with a hearing aid and began to learn lip reading his retested IQ was just double that of the original. It was a tragedy that he had to spend eight years before this simple test was done.

We saw a patient recently who had had an ascending viral meningitis who was diagnosed as aphasic, because they did not have a good otolaryngologist to examine and find there was because no otolaryngological examination had been made which would have disclosed a muscular paralysis. With simple muscular exercise and chewing gum, this woman is speaking audibly and quite well in less than 10 days. She learned to walk also, for she had a hemiplegia, and her total training time in the hospital was less than five weeks.

I believe that you have a problem in your profession, just as we have in general medicine. They say as far as those of us in internal medicine are concerned, between 80 and 90 per cent of all the patients that come to the internist's office are not problems of organic disease, certainly not acute infectious disease, but problems in the chronic disease field or in the psychosomatic field.

I say to our freshman class every year, if in all the time you spend with us you get only one thing, I will say your time has been well spent, and that one thing is this: If you can get the same inner satisfaction out of taking an old hemiplegic out of a wet bed, teach him to walk, dress and feed himself, talk, be dry, and to live outside the hospital and to get something out of his life if you can feel the same way inside as you now feel out of making a diagnosis of histoplasmosis or Kala-Azar, which you may or may not see in a medical lifetime, then your life is well spent, because these are the patients that you are going to see in practice.

If you do not know how to manage a hemiplegic, you will be a "barbital-potassium iodide" doctor. I speak feelingly because I was one for a long time; 17 years in internal medicine. I knew how insecure I felt when my hemiplegics came in. I had a technique. I put the blood pressure cuff on. They wanted to talk. I did not have time to talk. I had to take the blood pressure. If they talked I did not hear them. Before they had a chance to talk I would say "You are doing fine," and I would give them a mild sedative and hope they did not come back for a long time because I felt insecure.

I believe that you have the the same situation, in that your problems have changed with the antibiotics, and the young men coming along in your field are going to have to get the same inner satisfaction out of caring for the cerebral palsy child who has a problem in speech and hearing and also the

Ninety per cent of all hemiplegia patients can be retrained to perform all of the activities of daily living

old people. If young men can feel the same about their problems as they did in their younger days about a fresh, acute mastoid as a life saving, dramatic procedure then their professional experience is going to be richer and more profitable to their patients. These are the kind of patients that they are going to see.

I am frequently asked how much of rehabilitation is physical and how much is emotional? I always answer the question the same way. *If you do a day's work, how much do you do* with your right hand and how much with your left? In the first place, it depends upon whether you are right or left handed; second, it depends upon what kind of a job you have. If you know both of those factors at the end of the day you cannot tell. It is total.

We did a three-year follow-up on 146 patients who had been through our program in the early days, and we found that 55 per cent of them had had real emotional problems. Some of these problems we met, and some we failed to meet. I think we are meeting practically all of them now. It is silly to teach these persons to crutch-walk if they are not to go out of doors. The push has to be the satisfaction. It has to be the vocational, medical, physical, emotional; total.

We have here not only a medical problem but also an economic problem. If we do not do something about meeting this problem, by 1980, for every ablebodied worker in the United States there will be one individual beyond the age of 65, one chronically ill or one physically disabled worker, in addition to his dependent family. The economists tell us that the present situation is economically unfeasible.

We have a special responsibility in medicine and a special opportunity as well, for we have a tool in medicine, and especially in the programs for disabled people, that can build a firm foundation for international understanding. If we do not have that we cannot have peace.

My interest in this program began in 1949 during a mission to Poland. I went three weeks after Mr. Field disappeared at the airport, a very disconcerting time. I was sent by the United Nations as a consultant to help Poland set up a rehabilitation program. Poland was on her knees then; Warsaw was more rubble than city; the people were hungry, cold and bitter, and in a desperate situation.

When I left I thanked the Minister of Labor but he said, "Thank *you* for this mission. This has been a good mission. Not only have we learned something to help us with our disabled, but more than that we have learned that Poland and the United States have many common problems." He said, "You know, if we could just work on these together, one day maybe we could understand each other well enough to live in peace together in the world. That has to be our objective."

I said, "Those words were worth the mission to me; will you put them into writing?"

He said, "You will have them before you leave." As the train pulled out of this rubble pile, which was a station, a man in a greenish black suit stepped out from behind the rubble and handed me an envelope.

The doctor who took me about on that mission was the professor of Orthopedics of the University of Posnan, a distinguished physician. I remember the first morning that we went into his study. It was cold; they turn the heat on there by the calendar, not by the temperature, and we had a cup of tea. There was a magnificent portrait of a young physician on the wall, black hair, long white coat, stethoscope in his pocket. I said, "Prof. Dega, that is a wonderful looking young doctor. I hope I get to meet him."

He said, " You won't meet him; he is dead. He was in Warsaw during the uprising, and there was a child in the ghetto who got acute osteomyelitis in the late afternoon. Word came to this young physician through the underground. He knew that if the child was not operated upon that night, it would not survive. He went into the ghetto, and in the midst of the operation the Nazis came with machine guns and killed him, the patient, and everyone in the operating room."

Prof. Dega came to two meetings recently, the International Poliomyelitis Congress in Rome and International Society for the Welfare of the Cripples' Meeting in The Hague. Day before yesterday I received an official request from the Government of Poland applying for membership in the International Society for the Welfare of Cripples.

I have seen this happen in Israel, Greece, Korea, and all over the world in medicine. We have a common language. Osler said it simply in his early days of writing: "Medicine knows no nor has it ever known an international boundary." If a man's legs are off or if he is deaf or blind, the basic problem is the same regardless of his religion, politics, color, geographic

Good fitting and sound training in the use of prostheses are as important as the prosthesis itself

origin" I have never found any place in the world that you could not talk about these problems on common ground

I feel that we have not done enough throughout the world in this program We have spent millions of dollars in our aid programs, and I am afraid that sometimes we have forgotten people.

I ran across a little note in a medical journal last week that put this in high focus to me A little hospital medical society in Great Britain sent to the medical school in Afghanistan four boxes of pathological slides. They were so excited about

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

The meeting dates of the Sixth International Congress of Otolaryngology are again emphasized as May 5th through May 10th, 1957. The scientific program for the Plenary Sessions is now complete and is as follows:

CHRONIC SUPPURATION OF THE TEMPORAL BONE.

OPENERS: Marcus Diamant, Central County Hospital, Halmstad, Sweden—Anatomical Etiological Factors in Chronic Middle Ear Discharge.

Luzius Rüedi, Zurich, Switzerland—Pathogenesis and Treatment of Cholesteatoma in Chronic Suppuration of the Temporal Bone.

Horst Wullstein, Director, Otolaryngological Clinic, University of Würzburg, Germany—Surgical Repair for Improvement of Hearing in Chronic Otitis Media.

DISCUSSERS: A. Tumarkin, Liverpool, England; Juan Manuel Tato, Buenos Aires, Argentina; T. E. Cawthorne, London, England; Fritz Zöllner, Freiburg, Germany.

COLLAGEN DISORDERS OF THE RESPIRATORY TRACT.

OPENERS: Hans Selye, Director, Institute of Experimental Medicine and Surgery, University of Montreal, Faculty of Medicine, Montreal, Canada.

Introduction:

Michele Arslan, Padua, Italy—The Upper Respiratory Tract.

Leslie Gay, Physician-in-Charge, Allergy Clinic, The Johns Hopkins Hospital, Baltimore, U. S. A.—The Lower Respiratory Tract.

DISCUSSERS: Victor E. Negus, London, England; Branimir Gusic, Zagreb, Yugoslavia; Aubrey G. Rawlins, San Francisco, U. S. A.; Henry L. Williams, Rochester, Minn., U. S. A.

PAPILLOMA OF THE LARYNX.

Openers: Jo Ono, Tokyo, Japan—Etiology.

Plinio de Mattos Barretto, Faculty of Medicine, University of Sao Paulo, Brazil.

Diagnosis:

F. C. W. Capps, London, England—Therapy.

Discussers: C. A. Hamberger, Göteborg, Sweden; Pedro Hernandez Gonzalo, Havana, Cuba; Eelco Huizinga, Groningen, Netherlands; Albert von Riccabona, Vienna, Austria.

Applications to present voluntary papers are being received regularly and anyone wishing to present such a paper should make known his intentions prior to the deadline of Oct. 1, 1956.

The motion picture sessions, as well as the scientific exhibits, will prove to be outstanding in every respect. It is essential that anyone wishing to present an exhibit should make his intentions known immediately, as the final date for consideration is Aug. 1, 1956; applications to present motion picture films should be sent in before Oct. 1, 1956. Anyone planning to attend the Congress and who has not yet registered should do so immediately in order to obtain hotel registration priority.

For more detailed information pertaining to the Sixth International Congress please communicate with the General Secretary, 700 N. Michigan Ave., Chicago 11, Ill., U. S. A.

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Time and Place: Brazil, 1958 or 1959.

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RHINOLOGY IN CHILDREN

Resume of and Comments on the Literature for 1955

D. E. S. WISHART, M.D.,

be given routinely but only when necessary. Sulfonamide compounds, penicillin, and other antibiotics are ineffective in virus initiated colds, but are valuable in the treatment of complications of the common cold. It would not be advisable to prescribe these therapeutic agents routinely for the prevention of bacterial complications, especially since it is difficult to prognosticate the course pursued by the common cold in any given patient. Temporary relief from the discomfiture of nasal obstruction justifies the use of nasal medication on a rational physiological basis.

Since the common cold in infancy often tends to produce complications, it requires adequate attention. A soft rubber ear syringe may be used to clear the nasal passages of infants who have considerable nasal drainage. The bulb of the syringe is compressed before introduction into the nostril and then allowed to expand, a procedure that withdraws the offending nasal discharge. When the opposite nostril is allowed to remain open there is no danger from excessive negative pressure. When the infant cannot take the breast or bottle because of breathing difficulty, due to suppurative nasal blockage, the use of the syringe immediately before feeding time frequently enables the child to take nourishment.

Sanders[2] considers the child with persistent nasal symptoms as a very common but frequently unsatisfactory problem. It is usual for such a child to remain without accurate diagnosis; for there are many doctors who cannot make a nasal examintion, and there are also many otolaryngoloists who have neither the time nor the patience necessary to examine and treat children. All children with nasal symptoms should have a nose and throat examinaton that is thorough. Differentiating allergy from infection in all cases is necessary. A thorough nasal examination should be performed before removal of tonsils and adenoids. Removal of tonsils and adenoids should not be expected to clear up nasal symptoms in all patients.

Walsh[3] discussed vasomotor rhinitis. Allergic rhinitis by common usage refers to cases such as hay fever where the symptoms are acute, due to an antigen-antibody reaction which can usually be demonstrated by history or by skin tests. The

Parish makes several pertinent comments on the above letters. He points out that one letter clearly emphasized that overdosage in treating infants with any nasal decongestant must be avoided. He shows that the manufacturers of Tyzine indicated the possibility of soporific effects in infants and printed instructions that both the patient and the bottle must be upright for the administration, in order to avoid pouring the solution as a stream. His own extensive experience showed that no unfavorable effects had occurred when these precautions were followed.

Comment. It is difficult for a mother to carry out such instructions. It is practical, usual and easier to put drops in the infant's nose when it is lying on its back. Unless the dangers are printed in big print on the container, the mother will not read the manufacturer's caution. We, therefore, commend the remarks made by Greenstein and Friedman.

Tompkins and Macaulay[8], noting that the multinucleate giant cell of lymphoid tissue know as the Warthin-Finkeldey cell is a well-known finding in prodromal measles, considered that these cells might occur in nasal mucus. They demonstrated these cells in ten patients ranging from five days prodromal to the day of exanthem. Control preparations from patients with upper respiratory tract infections, allergic states, rashes, roseola, and rubella failed to show any comparable structures.

They describe the technique clearly.

Their own experience has been so encouraging that they recommend the method to their clinical and laboratory colleagues and are gratified to discover that the disease may be so simply and directly identified in the laboratory.

Schenck[9] stressed the importance of the plasma cell theory of antibody formation in the interpretation of the physiology of the lymphoid structures of Waldeyer's ring. The theory is that an unaltered bacterial cell is split by reticuloendothelial cells and polymorphonuclear leukocytes into chemically active antigenic molecules. These are then able to stimulate the plasma cells in the synthesis of gamma globulin containing the majority of humoral antibodies. These activities take place in lymphoid tissue.

The strategic position of the lymphoid tissues of Waldeyer's ring suggests that they initiate reactions to produce antibodies against organisms present in the upper respiratory tract and oral cavity.

The physiological variations in size of Waldeyer's ring appear to conform with immunologic requirements, further lending weight to the theory that the prime function of Waldeyer's ring is the formation of antibodies.

NASOPHARYNX.

Ireland[10], writing of pathological lesions in the nasopharynx, states that the patient may present himself with the following symptoms. *1*. Obstruction to nasal breathing. *2*. Postnasal discharge, purulent, mucoid or bloodstained. *3*. Unilateral or bilateral deafness. *4*. Involvement of various cranial nerves. *5*. Unexplained firm mass in the cervical lymph nodes. He discusses the pathological causes of each of these and briefly outlines the treatment.

The discussion is general in scope but has some particular application to the problems of children. Under nasal obstruc-

ions naso-antral polyp, inperforate choanae,
benign *naso-fibroma. He states that he does*
subject completely or discuss all the various
is purpose is to correlate specific pathological
e nasopharynx with the associated clinical

hensive pathological review should be read in

sented conclusions up to the present on naso-
fense mechanisms.

e of defense is the epithelium which is by its
and its covering of mucous secretion removes
bacteria. The pH of the nasal secretion is
the onset of local disease, and there is an in-
mucous secretion to cope with the irritant.
engthen this first line of defense, but it may
ental blockade of the nasal passages and filling
.

y of the epithelial defense mechanism is altered
yperventilation or loss of cilia due to trauma.

f sinus mucosa changes it from simple thin
ar epithelium to a thick membrane with much
ltration of phagocytic cells.

These cells constitute the second line of defense, but the swelling may increase and aggravate the situation by closing the sinus ostia. When this state of affairs has existed for a month or so the actively phagocytic neutrophiles and histiocytes are replaced by plasma cells and fibrocytes which *work together to encourage* healing.

The retropharyngeal and superior cervical lymph nodes are the third line of defense.

When the infection passes these barriers drainage into the cervical lymph ducts may occur with eventual drainage into the superior vena cava. This in turn can carry the infection via the right heart and pulmonary capillary bed to the perivascular and peribronchial lymphatics, which are the fourth barrier to systemic invasion by upper respiratory infection.

SINUSITIS.

A paper on sinusitis in children was presented by Wishart and Wallace[12] before the Canadian Otolaryngological Society. They did not pretend to give a complete treatise on sinustis in children, but instead they discussed the present attitude at the Hospital for Sick Children in Toronto towards sinusitis in these patients. Sinusitis is the common cold which has invaded a sinus; in children it is exceedingly common, the antrum being the sinus most frequently involved.

In the milder types the sinus is unable to perform its normal function, because an obstruction near the ostium so impairs the activity of the cilia of the lining membrane that they cannot evacuate the infection. When the trouble is more severe or of longer duration there is a puddle of discharge which is too heavy for the cilia to remove. If function can be restored in these cases, a cure results.

The nasal mucosa of a child with acute sinusitis rarely shows marked injection. Good inspection of the membrane is obtained by nasal shrinkage after a preliminary spraying with a mild vaso-constrictor. The use of the nasopharyngoscope is important and gives one an accurate picture of the nasopharynx and adenoid pad if present. X-rays of the sinuses are a help but should not be taken during an acute upper respiratory infection.

A great preventive of sinusitis is the natural cleansing of the nose. Many babies cannot get rid of the nasal secretions on their own, but placing the infant face down allows the secretions by gravity to fall to the front of the nose where the crying child will snort them out. Natural evacuation of the sinuses can be helped by treatment with vasoconstrictor drops. These drops must reach the middle meatus, and to do this the lateral-head-low position described by Parkinson is used.

Antrum lavage is a valuable procedure, and many antra that have been cloudy on X-ray over a long period will clear on one antrum washing; others require several washings to clear, and if the measure has failed then antrostomy is necessary. In performing an antrostomy a blunt Kerrison punch is used to bite out an opening, and great care is taken not to damage the inferior turbinate.

Acute ethmoiditis in a child is characterized by swollen, discolored eyelids and is often mistaken for an ophthalmic condition. The source of the infection is the anterior ethmoid cells, and unless this area is treated adequately, recurrences will happen.

The milder type of sinusitis is relatively free of exudate at times, especially in the summer months. Small doses of triple sulphonamides daily all winter help these children greatly by keeping them free from secondary infection. The more severe types are never free of exudate, and some of these patients require repeated lavage, or intranasal antrostomy, or sometimes the Caldwell-Luc operation.

The nose is an important physiological organ, and treatment of it should be goverened by good medical principles. Surgical technique employed in the treatment is very important, but it should be subservient to good medical evaluation.

Brown[13] says that in his experience the commonest cause of abdominal pain in children is chronic sinusitis. Evidence of chronic sinusitis as a frequent cause of abdominal pain is discussed under the headings of seasonal variance, presence of colds and other symptoms of sinusitis, diseases associated with both abdominal pain and sinusitis, and response to treatment of sinusitis.

Seasonal Variance. Comparatively few cases of abdominal pain are encountered in hot summer weather. In the fall, abdominal pain becomes a prominent symptom, and is also frequent in winter and spring.

Presence of Colds and Other Symptoms of Sinusitis. The author claims he has found usually sinusitis present in children with recurring abdominal pain, and children with sinusitis often complain of abdominal pain. The most common symptoms observed were anorexia, nausea, vomiting, head cold, cough, low-grade fever, fetor oris, photophobia, headaches and pain in the legs. Although other causes will produce each symptom, only sinusitis produces all of them. Signs of sinusitis in children with abdominal pain include circles and puffy under eyes, puffs beside nose, nasal obstruction, mouth-breathing, photophobia, sinus tenderness and cervical adenitis despite a previous tonsillectomy. The cervical adenitis is secondary to bacterial toxins from a sinusitis and the mesenteric adenitis may have a similar origin.

Diseases Associated with Both Abdominal Pain and Sinusitis. Many diseases are discussed which have a common organism producing the abdominal pain and nasopharyngitis or sinustis. Others have simultaneous associated symptoms. These illnesses include mesenteric adenitis, appendicitis, primary peritonitis, rheumatic fever, Henoch's purpera and some cases of so-called colic.

Response to Treatment of Sinusitis. The cause of the abdominal pain must be determined before treatment is commenced. One must rule out surgical and other serious conditions which occur in only 5 per cent of these cases. The large group which is secondary to upper respiratory infections respond well to the treatment of an active sinusitis. The nose is kept free by means of Isotonic astringent nose drops, or sprays followed by hot wet towels to the face after applying cold cream. This treatment is carried out three times daily, or more often if the nose is obstructed. The head-up position is used during sleep and Fowler's sitting-up position during working hours. When abdominal pain is due to acute sinusitis a penicillin injection is added to the above treatment.

To prevent recurrance of abdominal pain in children the treatment of chronic sinusitis twice daily should be carried on indefinitely.

The feature which makes sinusitis in children more important than the foci of infection is the development of pus under tension, which occurs in the confined space of a sinus.

Thompson[14] reported a fatal case of air-embolism following antral lavage.

The patient was a five-year-old boy who had the lavage done under general anesthesia. When the trocar was introduced there was some difficulty, and the operator had the pression that the instrument had gone on through and uck the postero lateral wall. The attempt to lavage with saline produced a very poor trickle. Afterwards air was blown into the antrum in the usual manner. There was some resistence to the flow of air through the antrum. Suddenly it was observed that the child had stopped breathing and was ashen grey. Resuscitation measures including cardiac massage failed to revive the patient, and at autopsy the child was found to have died from air-embolism introduced through a small puncture in the mucosa of the antrum opposite the site of entry.

In 1920 Gording was able to produce convulsions and death in rabbits by the insufflation of air into the maxillary sinus. Pang in 1952 quoted 58 cases with 23 deaths in humans.

Air embolism is a rare complication of injecting air into the antrum, but enough cases have been recorded to make it reasonable to inquire what is gained by this insufflation of air.

Since the fatality here reported the author has discontinued the insufflation of air and has never had cause to regret the decision.

He usually asks the patient to blow his nose gently, and this expels a few drops of the irrigation solution through the cannula. He considers there is no danger of self induced *air embolism* because there is also increased intra thoracic pressure.

DRUGS

Owings[15] reported on the control of postoperative adenoid bleeding with Adrenosem in the Germantown Hospital. Prior to using this drug their incidence was as high as 10 per cent of all cases.

Since using Adrenosem routinely there was only one case of adenoid bleeding in 102 cases plus three others which showed some bright red blood from the nose and mouth. These three cases were quickly controlled by a 5 mg. intramuscular injection of the drug.

Adrenosem checks bleeding from a capillary bed by correction of excessive permeability. It has no sympathomimetic action and appears to be non-toxic.

It can be given orally or intramuscularly. When given intramuscularly it is given shortly before operation.

Peele[16] reported on the use of Adrenosem in the control of hemorrhage from the nose and throat.

Adrenosem is a synthetic chemical. It is an oxidation product of epinephrine which is prepared as a stable aqueous solution by using sodium salicylate as a solubilizing agent.

Adrenosem has no sympathomimetic action, nor has it any effect on the blood-clotting mechanism. It exerts an increase in capillary resistance and a reduction of excessive permeability. The drug has no effect on large severed blood vessels or arterioles.

Adrenosem has a very high index of therapeutic safety, and there are no contraindications to its use at recommended dosage levels.

There is, however, evidence to show that the effects of Adrenosem are inhibited by the action of antihistamines, and these drugs should be discontinued for 48 hours before the administration of Adrenosem.

CONCLUSIONS.

The data assembled in this study seem to warrant the following preliminary conclusions. The routine preoperative use of Adrenosem in tonsil and adenoid surgery diminishes both the bleeding at the time of operation and the oozing in the immediate postoperative period. As a result of the reduced bleeding at operation fewer patients require suture of the tonsillar fossae for the control of hemorrhage. The incidence of primary postoperative hemorrhage may be reduced. Adrenosem is valuable in the active treatment of postoperative tonsil and adenoid bleeding and may reduce the incidence or obviate the necessity of postnasal packing or sutures.

In case of submucous resection of the nasal septum the of Adrenosem reduces the bleeding at the time of oper- on, in the postoperative period, and after removal of the nasal packing.

Preoperative Adrenosem reduces the bleeding following removal of benign inflammatory growths from the subglottic larynx and the operative field is thereby better visualized to determine completeness of removal.

Adrenosem is valuable in the control of hemorrhage from the choanal region of the nose seen in cases of hypertension, arteriosclerosis, or respiratory infections with or without local predisposing pathological conditions in the nasal cavity. The necessity of postnasal packing may be obviated by Adrenosem therapy in the majority of these types of cases.

Menger[17] reported on the use of parenteral estrogen in six cases of spontaneous epistaxis and ten cases of hemorrhage following adenoidectomy:

"The response in all cases was dramatic, and in only three cases was the length of time between the administration of the medicament and the cessation of bleeding over one hour. The consistency and rapidity of response rule out the possibility of automatic cessation and coincidence. Three possible mechanisms of action of estrogen are considered, but besides the passing evidence that estrogen may stimulate the release of thrombocytes, this study does not contribute any information toward the solution of the enigma. Even though the therapy at the present must be considered empirical, the effectiveness of parenterally given estrogen and the control of spontaneous epistaxis and bleeding following adenoidectomy offer strong evidence that merits serious consideration."

Harkins[18] made a study of the effects of vitamins K and C on postoperative bleeding after adenotonsillectomy.

Previous investigators had not been able to show any benefit as regards to hemorrhage where vitamin K was used alone.

Since vitamin C is of importance to healing and vitamin K is concerned with the prothrombin level a combination of the two vitamins was used in this study.

The study was made on a series of 200 children whose ages ranged from five to 13.

One hundred of the children received vitamin C and vitamin K for five days before operation and for five days after operation.

Fifty of these were allowed acetylsalicylic acid postoperatively and the other 50 were denied salicylates in any form.

The other 100 patients constituted a control group who received no vitamin C or vitamin K, and were allowed acetylsalicylic acid during their convalesence.

The results showed a reduction in the incidence of postoperative bleeding (both primary and delayed) from 14 per cent to 3 per cent. The frequency of postoperative hemorrhage was not increased in patients who were allowed acetylsalicylic acid if they also had been treated with both vitamin C and vitamin K. No toxic symptoms were noted which might be attributable to the vitamins.

Although careful bleeding and clotting time studies were made in all patients, these tests failed to indicate impending bleeding in the children who did suffer postoperative hemorrhages.

ALLERGY.

Van Alyea[19] has discussed the management of allergic sinusitis and stresses the need for close co-operation between the allergist and rhinologist.

He made several interesting observations which are worth noting. Contrary to general belief, polyps do not originate from within ethmoid cells. They commonly arise from the margins of sinus ostia, the crest of the ucinate process and anterior face of the bulla ethmoidalis; therefore it is not necessary to interfere with the ethmoid cells in the operation of polypectomy.

Allergic sinusitis can be roughly calculated into five groups:

1. Acute suppurative sinusitis on an allergic basis.

2. Subacute sinusitis maintained by structural drainage barriers and allergic edema.

3. Acute and subacute cases with drainage difficulty due to polyps.

4. Chronic sinusitis with blockage of outlets by edema.

5. Chronic sinusitis with or without polyps which has been subject to one or more radical operations.

Treatment in addition to careful allergic management is the same as in non-allergic cases with the goal ever being the establishment of adequate drainage.

Radical procedures are seldom advisable because allergic membranes removed are replaced by membranes also allergic and the patient's symptoms temporarily relieved may return along with others and perhaps be more pronounced than previously.

The *Jour. A. M. A.*[20] was asked if allergy to milk, in addition to bloating, also causes an increased post-nasal discharge. They replied that when a patient attributes mucus to milk, allergy to milk often is responsible, but goes on to show the care which must be taken before deciding that an allergy is due to a food.

Segard, of the Wisconsin Alumni Research Foundation, took exception to the above opinion stating, milk does not cause or give mucus or phlegm as commonly called. The mucus is already there in the oral cavity and the pharynx. Milk and other substances containing buffers neutralize the tenacious mucus. It then loses its tenaciousness and can then be expectorated. Speakers, actors, preachers, and others use milk to clear their throat of mucus. This reaction has nothing to do with disease. This reaction can be secured by the average toothpaste; it has nothing to do with allergy.

Craft[21] discussed the relation of allergy to the tonsils and adenoids. The results of adenotonsillectomy are at times disappointing. This is especially common when the patient suffers from a nasal allergy.

The presence of a nasal allergy must be detected by a careful history and thorough examination. It would seem logical to treat the allergy as well, before deciding that removal of tonsils and adenoids is likely to be helpful.

Another relationship occurs in the allergic individual who has developed a bacterial sensitization to organisms harbored in the tonsils or adenoid. This turn of events can be suspected from the history and bacteriological studies, and can be confirmed by intradermal skin testing of the patient with extract obtained from the infecting organism. The results of removal of tonsils and adenoids in this group along with autogenous vaccine is indicated.

A third observation in allergic patients is an increased tendency for the "growing back" of tonsils and adenoids after removal. These regrowths of lymphoid tissue occur especially in the nasopharynx of allergic patients. When the allergy is adequately treated pre-operatively the incidence of regrowth can be reduced from 27 per cent to 3 per cent.

CONGENITAL ABNORMALITIES.

A very lucid description of the mechanism of asphyxia in bilateral choanal atresia is given by J. V. D. Hough[22]. He demonstrates by means of drawings and X-rays the inspiratory and expiratory efforts of the infant when both posterior nares are obstructed. On inspiration the tongue and soft tissues of the floor of the mouth are sucked up and back to the hard and soft palates, producing a seal which prevents entry of air into the trachea. The epiglottis and soft tissues of the pharynx are sucked together, producing almost a drawstring effect. The expiratory effect can be explained by the ballooning produced by trapped air in the nasopharynx. The expiratory squeeze of the lungs and action of the muscles of respiration produces considerable air pressure on expiration. This builds up rapidly in the pharynx and nasopharynx and forces the soft palate down and backwards againt the tongue, thus producing a soft tissue seal. The pharynx and nasopharynx become greatly dilated. This seal must be broken if life is to be maintained.

The author maintains that the diagnosis of choanal atresia is easily made once the condition is suspected. The infant has intermittent dyspnea with variable degrees of cyanosis from birth. There is retraction of the suprasternal intercostal and infrasternal areas on inspiration; also, there are prolonged efforts at expiration with excessive use of the secondary muscles of respiration. Attempts to feed the infant increases the dyspnea. The cry has a normal sound, and crying visibly relieves the distress of breathing. Closure of the mouth produces immediate cyanosis. Examination of the nose shows considerable mucus lying in each fossa. It is impossible to insert a catheter or blunt probe through the nose into the nasopharynx. Iodized oil dropped into the nose cannot be traced to the pharynx with a roentgenogram.

The first thing to be done in the way of treatment is to break the seal between the palate and the tongue thus permitting better respiration. This is accomplished by passing a plastic tube through the mouth into the stomach. The tube also allows the feeding of the infant with safety. It may be necessary to tape a second catheter in the mouth so that it rests on the back of the tongue, to further break the seal between the soft palate and tongue.

Surgical correction of the defect may now be carried out, and this should be done as soon as possible. The author enumerates several surgical procedures but discards them all for a simple surgical technique which has given good results. Under endotracheal anesthesia a ball-tipped metal probe is inserted along the floor of the nose, close to the septum, until it rests against the atresia. The depth is measured from the columnella. A guarded trochar is passed into the nose to the measured length, taking care to reach the medial and inferior area of the atresia. This area is then penetrated, and the posterior wall is protected by a small laryngeal mirror in the nasopharynx.

Following the trochar, a slightly curved Van Alyea antrum cannula is pushed through the penetrated area to enlarge it slightly. Further enlargement of the orifice in the atresia is accomplished by infant urethral dilators. The penetration and dilatation procedures are repeated in the opposite nares, and so both are made to communicate with the nasopharynx. Two polyethylene tubes the size equal to 14-E are inserted through the nares into the nasopharynx to the back of the soft palate and are secured anteriorly by a safety pin which is taped to the face. The tubes are left in place for three weeks or longer, and during this time they are kept clean by suction and may be replaced if necessary. Local medications in the nose are avoided, but antibiotics may be given systemically to prevent infection. After removal of the tubes the openings may tend to close. If this is so the proper procedure is simple dilatation, as before, when considered necessary.

This seems to be a very practical, simple, and sensible way to treat bilateral choanal atresia in the newborn.

TONSILS.

Moore[23] made some suggestions with respect to general tonsillectomy. Proper preparation of the child is essential. In this regard there is a very clever record by Mercury Childcraft, called "Peter Ponsil Lost His Tonsil". Measures should also be taken to be sure that the parent can be with the child after the operation, so that he does not have the feeling of being abandoned to strangers.

There has been some dissension regarding preanesthetic sedation for children. Heavy enough sedation to reduce the incidence of crying increases the hazards of the anesthesia. It is probably safe to give atropine to most children.

There is also some dissension about the use of endotrachial anesthesia in children. Many experts feel that it is not necessary and may produce postoperative laryngeal edema.

Peritonsillar injection of normal saline containing 6 minims of 1-1000 adrenalin to the ounce will reduce the amount of bleeding during the operation even in adults. These injections are contra-indicated in children under two, diabetics, and patients with coronary sclerosis. It is also inadvisable where anesthesia is being produced by cyclopropane.

There seems to be mounting evidence that the incidence of secondary hemorrhage is greater when acetylsalicylic acid is used.

The use of efocaine as a long acting local anesthetic to reduce postoperative pain has been of questionable value, and there are reports that there is considerable danger involved. There is no clear evidence that Cortisone or ACTH produce more bleeding, but some experiences suggest that it might be wise to withdraw the drug for four or five days before the operation is done.

A short period of pre-operative Cortisone, or Hydrocortisone on the other hand, may help protect a thymicolymphatic child from the hazards of surgical trauma; as well, the intravenous use of Hydrocortisone has been found useful in combating the hypotension of surgical shock.

The *Jour. A. M. A.*,[24] asked regarding the relation of diseased tonsils to tonsillectomy, replied that tonsillectomy was not more frequent in families that are in the higher income bracket and that, since virulent organisms are found in tonsils soon after birth, almost all tonsils should be considered diseased.

The Southeast Metropolitan Regional Hospital Board[25] has approved plans for an inquiry into the value of tonsillectomy. At Farnborough Hospital, Kent, England, and perhaps other hospitals in that area, 1,000 children on the tonsils waiting list will be chosen for the trial: 500 will have their tonsils removed and 500 will not. The health of the two groups in the succeeding year will then be compared.

The *Lancet*[26] comments as follows on a study by McCorkle, Hodges, Badger, Dingle and Jordan, which appeared in the *New England Journal of Medicine,* 1955:

"The indications for tonsillectomy may seem to be based more on intuition than on reason. Evaluation of the results, by different methods of assessment, has already cast serious doubt on the value of the operation. Among the lexicon-like list of reasons for undertaking it, repeated respiratory infections (and what child does not have them?) has been well to the fore. Now McCorkle, et al., have made a painstaking assessment of the relation of tonsillectomy to the incidence of such infections. The study can be criticized at only one point, the small numbers concerned; but the 230 children studied were observed for up to five years, during which the mothers kept a daily health record, field workers paid a weekly visit, and physicians assessed all illnesses with the help of extensive bacteriological investigations."

The results are presented under two headings: under the first heading "the age-specific rate of infection" of two groups of children, tonsillectomised and not, before the period of observation began, is compared. Under the second heading is shown "the age-adjusted rate of infection" in a group of 26 children who, because they had a higher than expected rate of infection, were operated upon during the period of observation. McCorkle, et al., state: "Comparison of these data requires the use of either age-specific or age-adjusted rates, because the incidence of common respiratory illness changes with age, and post-tonsillectomy experience is heavily weighted with older children." The outcome of this study was that the operation had no effect at all, and the 26 children with a higher than expected rate of infection continued unchanged at the higher rate after the operation.

The great care with which this study was conducted suggests that, though the numbers were small, the results would be the same whatever the total.

Rhoads, et al.,[27] reported on a clinical investigation undertaken to throw light on the question of: *1.* The presence of a transient bacteremia after tonsillectomy that is similar to that observed after tooth extraction and its prevention by preoperative treatment with penicillin and other antibiotics; *2.* The amount of information regarding the bacterial content of the deeper lymphoid tissue of the tonsils given by cultures from the surface of the throat; and *3.* How antibiotic therapy affects the bacterial content of the deeper structures of the tonsil.

summary and conclusions are as follows:

d cultures taken just after tonsillectomy were positive 3 per cent of a group of 68 patients who received no otic therapy prior to tonsillectomy. The incidence of emia was reduced to 5.9 per cent in a group of 20 who received penicillin in a daily dose of 600,000 000 units intramuscularly for four to ten days prior sillectomy. The incidence of bacteremia in a group patients who received 600,000 to 800,000 unit doses this dose for children) of procaine penicillin 12 to 18 nd one hour prior to tonsillectomy, or in a small group of patients receiving 900,000 to 1,200,000 units aily for five to seven days prior to operation, was ced below that of the control group. Beta hemolytic occi were obtained from blood cultures four times, occi once, alpha hemolytic streptococci (green forming) Gamma a-hemolytic streptococci 28 times, and a combination of Beta hemolytic streptococci and Gama a-hemolytic streptococci twice. The need for several days preoperative treatment with penicillin to prevent post-tonsillectomy bacteremia is obvious.

In the control series of 68 patients who had no antibiotic treatment immediately preceding tonsillectomy, Beta hemolytic streptococci were present in 57.4 per cent of the cultures of the excised tonsils, although these micro-organisms were found in only 28.26 per cent of throat cultures taken just before the operation. Among patients receiving penicillin in simple doses only the day before and the day of tonsillectomy, 31.03 per cent had Beta hemolytic streptococci in the excised tonsils, although these micro-organisms were not present in the throat cultures taken just before tonsillectomy. Beta hemolytic streptococci were found only once in the cultures from the excised tonsils of persons receiving penicillin intramuscularly each day for four to ten days prior to tonsillectomy. Most of the gram-positive micro-organisms except micrococci and Gaffkya tetragenus were greatly reduced in number by penicillin administered intramuscularly, but gram-negative micro-organisms, such as Klebsiella pneumoniae, aerobacter aerogenes, and Escherichia coli, were found in increased numbers in cultures from the throats and excised tonsils of these subjects.

Ferguson, McGarry, Beckman and Broder[28] report that in a four-year-old girl immediately after dental extraction, tonsillectomy and adenoidectomy, a massive subcutaneous surgical emphysema developed and rapidly extended to produce a pneumomediastinum, pneumoperitoneum and a left tension pneumothorax. Recovery was rapid after establishment of underwater closed catheter drainage of the pneumothorax.

The mechanism of the air entry in surgical emphysema following tonsillectomy has been much discussed in the literature. Some authors favor the tonsillar bed as the site, others favor the intrapleural or extrapleural route, and yet a third school of thought claims that pulmonary interstitial emphysema is the initial lesion, and results from respiratory obstruction.

Macklin in his experiments showed that by over-inflation the alveoli can be made to rupture, resulting in a leak of air into the interstitial tissues of the lung. The air may then travel along the walls of the pulmonary blood vessels in artificial channels which it has dissected for itself to the root of the lung and from there into the mediastinum. This may further spread to give extensive pneumomediastinum and even a pneumothorax.

In this particular case the authors believe that the ragged tonsillar beds permitted air to enter the tissue planes during post-operative swallowing and coughing. Operative hemorrhage no doubt led to more trauma to the tonsillar fossae than usual, and this coupled with a moderate amount of respiratory obstruction resulted in the air extravasation, the air traversed tissue planes in the neck and extended into the mediastinum and retroperitoneal area. Rupture of a mediastinal bleb produced the left tension pneumothorax, and a similar sort of event must have allowed air to enter the retroperitoneal space.

Barton and Roman[29] described an endotracheal technique for adenotonsillectomy. The requirements of ideal tonsillectomy anesthesia are that it be safe, not too unpleasant, and that it allows proper working conditions for the surgeon.

To achieve these ends the authors devised three sizes of endotracheal tongue blade which can be used with the Crowe-Davis or the McIvor mouth gags. The blades are constructed as a conduit through which the endotracheal tube can be threaded. The device allows endotracheal intubation without interfering with the surgical field for any kind of otolaryngeal surgery.

They feel that this apparatus overcomes all the objections [illegible] to the use of Pentothal techniques without intubation. They further feel that the technique of endotracheal anesthesia permits the use of heavy premedication or the preoperative use of rectal Pentothal to minimize the psychological shock of the operation.

Comments: At the Hospital for Sick Children in Toronto, we still think that open ether anesthesia is the safest for tonsillectomy in children. We avoid premedication because it prolongs the return of a vigorous cough reflex which is the child's chief protection against the sometimes serious effects of postoperative aspiration of blood or vomitus.

PREMEDICATION AND GUILLOTINE TONSILLECTOMY.

N. G. P. Butler[illegible] believes that methylpentynol is a very unsatisfactory sedative for children prior to tonsillectomy. He argues this from his own experience and also from his reading of Dr. C. M. Rendell's article in the *Brit. Med. Jour.* of Dec. 11, 1954.

Only two children out of 56 admitted liking the preparation; the remainder disliked it intensely. He agrees with Dr. Rendell that in about a tenth of the cases the induction was delayed by vomiting the elixir. The ward staffs actively disliked the menthylpentynol on account of the almost universal postoperative restlessness and lack of cooperation that accompanied its use. Whereas the non-drug-sedated child is soon completely "round" after the operation and after suitable explanation and reassurance will lapse into quiet sleep, a child who has had methylpentynol is still confused and incapable of appreciating the situation, and thus reacts accordingly.

R. J. James Hodges also writes that the evidence in Dr. Rendell's article does not substantiate her favorable conclusions.

D. B. Halstead writes that any premedication swallowed gives increased gastric motility and increased risk of vomiting and its sequelae; any morphine derivative depresses respiration and makes it difficult to obtain the required depth of anethesia, and most children object more to rectal injections than to a gas tactfully given. All the barbiturates make the children restless and difficult to control postoperatively, unless there is a nurse for each patient. He remains convinced that the best and safest premedication is atropine 0.65 mg (by injection) and nothing else.

Gusterson[31] writes that in his opinion the elixir of "oblivion" as put up by Schering is of very great value for children. He has not met a single incident of vomiting in 500 cases. The oblivion should be given one hour before induction and followed by atropine half an hour later. He is preparing a film which he hopes will be convincing.

Methylpentynol is now being prescribed fairly widely as a hypnotic, and as a means of allaying apprehension, but there are relatively few reports of overdosage. The following case report by I. M. Brown and R. A. Ellis, describes recovery after ingestion of approximately 10 g. of methylpentynol in the form of a proprietary elixir ("oblivion") and this appears to be the largest overdosage followed by recovery.

A woman aged 39, weight 7 st. 8 lb., was admitted to hospital on Oct. 29, 1954, having taken approximately 5 oz. (150 ml.) of a proprietary elixir containing 250 mg. of methylpentynol per drachm (70 mg. per ml.) There was a history of previous mental illness, necessitating admission on a voluntary basis to a mental hospital in July, 1954, for a period of ten days. Since that time the patient had been depressed at intervals, and had threatened to commit suicide.

She had drunk the elixir about mid-morning and was admitted to hospital at 3:30 p. m. She was then deeply asleep but responded faintly to pressure on the supraorbital nerve. Her general condition was good; respiration 20, pulse 72, blood pressure 130/80. Her pupils were normal in size and reacted briskly to light.

On the assumption that absorption was probably complete, stomach lavage was not carried out. General supportive treatment was instituted and methylamphetamine hydrochloride 30 mg. was given intravenously every half-hour. At 6:30 p. m. she was less deeply asleep, and half opened her eyes when spoken to. Her general condition was good, respiration 21, pulse 72. By 8:30 p. m. she was half awake and making attempts to speak. At 10 p. m. she was able to speak and to answer questions, although still drowsy. At this point the methylamphetamine hydrochloride was discontinued.

During the next day she was somewhat drowsy, but rational and capable of answering questions. By the morning of Oct. 31 she was fully recovered and later consented to have further mental hospital treatment as a voluntary patient.

The single fatality on record is thought to have followed the ingestion of 4.5 to 6 g.

Gusterson[33], anesthetist, Worthing group of Hospitals, Sussex, England, has written a long useful article describing the management of children about to undergo tonsillectomy. The entire article is worthy of study because it contains many practical suggestions. He summarizes his paper thus: It is important to avoid emotional trauma in children admitted to hospital for tonsillectomy, especially when they are under five years of age. Simple points in the management of children during their stay in hospital are outlined. The disadvantages and dangers of using barbiturates for preoperative sedation are outlined. The use of methylpentynol in a flavored elixir preoperative sedation is described. A follow-up to 100 cases showed no difference between the emotional response of those children sedated with pentobarbitone and those receiving methylpentynol. Methylpentynol has a great safety margin and none of the disadvantages of the barbiturates.

W. N. Rollason[34] briefly states seven combinations for tonsillectomy and adenoidectomy in children and the reasons for discontinuing them. While the ideal premedication and anesthetic technique for guillotine tonsillectomy in children has yet to be found, syrup of chloral correctly given, followed by nitrous oxide, oxygen, and trichlorethyline, correctly administered from a McKesson machine is at present, he thinks, the nearest approach to this ideal.

E. H. M. Foxen[35] writes that he is surprised that guillotine tonsillectomies are still being performed in large numbers in the second half of the Twentieth century (circa 1955).

He mentions how Waugh found 143 cases that had already had their tonsils guillotined, some of them as often as two or three times, and concluded that the cause of the failure lies not with the operator but with the type of operation. He voices his agreement with Waugh.

D. Browne[36] agrees with Foxen in his protest against the extraordinary inefficiency of the present removal of tonsils and adenoids in Great Britain.

H. B. C. Sandiford believes that the key to the performance of good and bad tonsillectomy is anesthesia.

J. C. Campbell[39] assures Foxen that it is not just the guillotine method which is responsible for tonsillar remnants, and damage to the faucial pillars, soft palate, etc. If failures occur either the operator has not been taught correctly or is inept. To reverse Mr. Miles Foxen's quotations, surely "the cause of failure lies not with the operation but with the operator."

R. Ellison[37] argues that with proper skill and using a hemostatic guillotine in the majority of children, guillotining is as good as dissection.

J. F. Neil states that the last sentence quoted from Waugh is nonsense. The complete removal of a tonsil with a guillotine is perfectly possible.

Forster[38] is convinced that the most successful instrument to apply in the many cases suitable for its use is the modified double-bladed hemostatic Tonsillatome of LaForce.

Campbell and Smith[39] state that the correspondence which had recently appeared in the *British Medical Journal* (abstracts of this correspondence are printed in this "Tonsil" section) would lead one to suppose that the operation for removal of tonsils and adenoids in children by guillotine and curette under ethylchloride anesthesia was a thoroughly unsatisfactory procedure. This conclusion is entirely at variance with their experience.

They examine every detail of this procedure very fairly and terminate a good paper with the following summary and conclusions:

Guillotine tonsillectomy and curettage of adenoids carried out by an experienced surgeon, under ethylchloride anesthesia administed by a competent anesthetist, is a satisfactory operation. The procedure, as carried out by them is described in some detail, emphasis being laid on the necesssity for a considerable induction of anesthesia. Those who have recorded their objections to the operation base their opinions on an experience of inexpert anesthesia, usually too light. Contrary to widespread belief, the operation requires an anesthesia of considerable depth. The competent anesthetist who is aware of the dangers of ethylchloride, can employ it safely. A recent series of over 12,000 cases without a death is recorded, as contrasted with one of some 25,000 dating from the first quarter of the century in which there were eight deaths.

Crooks[40] carrying on a four year's tradition at the Hospital for Sick Children of dissection of tonsils in all cases, says that he cannot be expected to support the guillotine operation. He requires ten minutes of deep anesthesia, capable of extension exceptionally, for each tonsil and adenoid operation. He is horrified at the frequency of long lateral adenoid remnants causing ear trouble in his patients. More time and care are necessary. Ether anesthesia following ethylchloride induction is as safe and pleasant as ethylchloride alone, and, after all, what is the hurry?

Mawson and Cartwright[41] criticize several details in the article by Campbell and Smith. By contrast; they believe that the essential safety condition of the guillotine operation necessitates light anesthesia; they point out the danger of turning the child on its side after the removal of the tonsils and a leisurely adenoidectomy. They state that the guillotine operation has been abandoned because premedication was considered generally unsafe before guillotining children in large numbers; and finally because modern attention is becoming focussed more on the adenoidectomy and less on the tonsillectomy. Adenoidectomy is far more difficult to do well, and the penalties of an incomplete and inexpert removal are greater, since it is possible to cause permanent deafness through damage to the Eustachian tubes, or by failing to remove a cause of Eustachian obstruction and middle ear infection.

TONSILLECTOMY AND ADENOIDECTOMY AND DEAFNESS.

The *Jour A. M. A.*[12] was asked whether the removal of the tonsils would improve the hearing of a four-year-old child apparently hard-of-hearing, and replied that in view of the history given surgical removal of the tonsils and adenoids appear indicated.

Crowe[13] replied to a query regarding tonsils and hearing as follows:

"Our impression in the late 1930's was that the number of adults with impaired hearing could be reduced by 50 per cent if children in the primary grades of our public schools were examined with an otoscope and an electric nasopharyngoscope and properly treated. The routine procedure throughout the country at that time was to test the hearing and send a note to the parents. For financial reasons, often nothing was done for the child. In 1939 we examined with the greatest care 1,365 school children who lived in the Eastern Health District of Baltimore. The hearing of each child was tested on several different occasions in a soundproof room at the Johns Hopkins Hospital. Sixty-seven, or about one in 20 of these apparently healthy school children were found to have impaired hearing for all tones, and some difficulty in understanding spoken words. Many more had lesser degrees of impairment. This study was interrupted by the war.

"A second study of 5,428 third grade school children was begun in November, 1948, and terminated five years later. Screening tests showed that 582 (10.7 per cent) had some impairment of hearing. The hearing of the remaining 4,846 children was good. It is interesting that in this antibiotic era only seven of these 582 children had discharging ears. Recurring attacks of otitis media and chronic mastoiditis were common causes of impaired hearing before the days of sulfonamides and antibiotics. In the second and third decades of the present century, ear and sinus infections were more frequent than noted above, and I think for that period the 50 per cent prevention figure was not too high."

Barton[44] protested to the *Jour. A. M. A.* that Brown was misquoted or misinformed when he wrote that "Deafness, recurring otitis media, and mastoiditis are rarely improved by adenoidectomy."

Brown[45] replied to the protest as follows:

"In disagreeing with the above statement, Dr. Barton is undoubtedly voicing an opinion generally held; however, my article and *The Journal's* abstract of it would have served little purpose if they had not mentioned the fallacies of the prevailing belief. Many outstanding men believe that progressive deafness, recurring otitis media, and mastoiditis usually are caused by chronic sinustis, and that adenoidectomy not only fails to improve these conditions but may predispose to them. In the original article, 'Harmful Sequelae of Adenoidectomy in Children with Chronic Sinusitis,' *Arch. Pediat.*, 71:233, 1954, there are 64 references and many quotations regarding the protective value of adenoids against the purulent secretions of sinusitis in the production of the above conditions by sinusitis.

"The adenoid protects not only the upper but also the lower respiratory tract. Kaiser's controlled study, now a classic, has shown that removal of tonsils and adenoids increases the likelihood of lower respiratory tract symptoms. As my article states, 'these complications are seen only in children with sinusitis whose protective barrier, the adenoids, had been removed.' It seems neither fair nor open-minded for one to protest a statement appearing in an abstract when he has not read the original article to determine how the conclusion was derived."

Replying to a query regarding the use of radium in the nasopharynx the *Jour. A. M. A.*[46] replied:

"This form of therapy is still very popular, and is producing the desired results in the usual percentage of cases. So far there is no definite evidence to show that carcinoma can result as a late radiation sequel. Some reports show patients followed as long as 30 years without serious pathological consequences."

POLIOMYELITIS AND TONSILLECTOMY.

A report on the relationship, if any, of poliomyelitis and tonsillectomy[47] has been issued by the Medical Research Council Committee on inoculation procedures and neurological lesions. This report is so detailed and so carefully composed that it should be studied in the original.

It is divided into two parts: one dealing with recent tonsillectomy and the other with remote tonsillectomy. Little more than the summaries of these parts can be given here.

1. *Recent Tonsillectomy.* Since early in 1951 all medical officers of health in England and Wales have submitted to the Medical Research Council a routine report on each person notified in their areas to be suffering from poliomyelitis. The reports stated whether the patient had undergone tonsillectomy within 12 months before the onset of symptoms and gave dates where relevant. Patients reported to have had their tonsils removed within three months before the onset of poliomyelitis were selected for special investigation, and one of three investigators visited the medical officer of health who notified the case, the physician who was in charge of the patient in hospital, and, in many instances, the patient's general practitioner, and made a detailed record. This is a report of the information obtained about all patients in England and Wales up to the end of 1953, said to have had their tonsils removed within the three months before the onset of poliomyelitis.

Between March, 1951, and December, 1953, complete reports were received of 103 children up to 18 years of age who developed poliomyelitis within 91 days after tonsillectomy.

Sixty-one (59 per cent) had symptoms one to 21 days after tonsillectomy. The concentration was most evident in the bulbar and bulbo-spinal groups where 44 (79 per cent) occurred one to 21 days after operation, compared with 38 per cent of the spinal cases and 31 per cent of the non-paralytic cases.

Most cases occurred in the principal epidemic period, but the same pattern was seen in epidemic and non-epidemic seasons.

Tonsillectomy, as practiced at present, added relatively few to the total number of cases of poliomyelitis reported during the study, but tonsillectomy should continue to be restricted in any area where the disease is unusually prevalent.

2. *Remote Tonsillectomy.* In the summer of 1951, an investigation was made to determine the importance of the etiology of poliomyelitis of activating agents such as physical activity illness, injury, injections, and surgical operations, including tonsillectomy and dental operations. For all agents except tonsillectomy the inquiry was restricted to three months before the onset of poliomyelitis, but for tonsillectomy a record was made of operations carried out at any time before the onset of symptoms. The investigations were made in Cardiff, the administrative county of Devon, East Anglia, Exeter, Leeds, parts of the administrative county of London, Manchester, and Oxford, and as only a few cases were obtained in 1951, the inquiry was continued until the end of 1952.

Between mid-1951 and November, 1953, records were obtained in selected areas of England and Wales of the history of tonsillectomy at any time before the onset of paralytic poliomyelitis in 51 patients under five years of age and 203 patients five to 15 years of age. Histories were similarly obtained for the same number of matched controls. Only two of the patients and none of the controls under five years of age had had their tonsils removed.

In the five to 15 year age group, 72 (35 per cent) of the patients had undergone tonsillectomy compared with 44 (22 per cent) of the controls. The differences between patients and controls were greatest in the group with bulbar paralysis, intermediate in the group with bulbo-spinal paralysis, and least in the group with spinal paralysis.

In the bulbar and bulbo-spinal groups, most patients had had their tonsils removed at least one year and often five or more years before the onset of poliomyelitis.

The results indicated that persons whose tonsils had been removed were more likely to develop the bulbar form of poliomyelitis than those who had not had the operation, even if years had elapsed between the removal of the tonsils and the onset of poliomyelitis.

Fraser[48] doubts whether it is fair to contrast a group of tonsillectomised children with a control group of average children. The former presumably have a poor resistance to upper respiratory infections, and it would, therefore, be reasonable to expect a more severe attack of poliomyelitis in this group than in a group of average children.

Asked whether it is advisable to perform tonsillectomy and adenoidectomy on children taking the Salk vaccine, the *Jour. A. M. A.*[49] replied: "On the basis of available evidence that tonsillectomy may have a provocative effect on poliomyelitis, it would seem wiser that a child who is to have a tonsillectomy should be given the course of the vaccine before the tonsillectomy is undertaken."

The Jour. A. M. A.[50] was asked when tonsillectomy and adenoidectomy should be performed in relation to administration of Salk vaccine and replied:

"If it is desired to derive what protective influence vaccination may provide before tonsillectomy, then the period when *the full protective effect can be anticipated from primary* immunization would be 14 days after the second dose, even though it has been shown that in the majority of individuals antibody developes 14 days after the first dose. The interdiction of elective surgery in relation to the administration of poliomyelitis vaccine was applied in some areas during the field trial period. Elective surgery, and more specifically tonsillectomy, should not be performed during periods of poliomyelitis prevalence, but this need not deter tonsillectomy if vaccination is administered when poliomyelitis is not prevalent. It would be hoped that under ideal circumstances a course of poliomyelitis vaccine could be completed before tonsillectomy to take advantage of whatever immunization has resulted *from the vaccine. One should not tempt fate by advocating* tonsillectomy during the poliomyelitis season even in vaccinated children. Efficacy of vaccination was demonstrated under normal circumstances, and not under circumstances of the increased hazard that pertains as a result of tonsillectomy during poliomyelitis prevalence."

MOUTH AND THROAT INFECTIONS.

In diphtheria the antitoxin is very effective in neutralizing the exotoxin formed by the C. diphtheriae, but toxin continues to be produced until the bacteria cease to multiply. All the available antibiotics have some bactericidal effect on the organisms, both locally and systemically. Beach[51] and his co-workers studied the effect of erythromycin on the clinical course and bacterial flora of the nose and throat in acute cases of diphtheria, and in chronic carrier state. In 1953 and 1954 they treated 103 cases, and of these selected 49 for study. The other cases were discarded from the study because of previous medication before coming to hospital.

The 49 patients all had one or more positive nasal or throat cultures for C. diphtheriae with subsequent confirmation by virulence tests. They were treated with erythromycin and diphtheria anti-toxin. All patients on the pediatric service of their hospital who were found to be diphtheria carriers were treated with erythromycin alone. Patients classified as carriers were completely asymptomatic, had no physical findings to suggest active infection, had negative Schick tests and harbored virulent C. diphtheriae in their oropharynx.

Cultures for C. diphtheriae were obtained daily on most patients for seven days, and as long thereafter as necessary until two consecutive negative cultures were obtained. Levels of erythromycin in the sera of 28 patients were determined at intervals during the course of the treatment. Any toxic effect of the drug was noted.

It was found that C. diphtheriae was eradicated in all patients after completing treatment. The average time was two days in the active cases and three days in the carrier state. One patient died from toxic myocarditis. In the treatment of diphtheria erythromycin appears, on the basis of this study to be the most promising antibiotic to date.

Erythromycin is advocated as an adjunct to and not as a substitute for antitoxin in the treatment of acute diphtheria.

The *Jour. A. M. A.*[52] was asked for diagnosis and treatment of tenacious pseudomembrane formation over the adenoids which was unresponsive to chemotherapy: 27 cases with this condition had been seen, three of them in children. The reply was that mononucleosis and lymphatic leukemia should be considered, but emphasized that diphtheria was a strong probability in spite of the negative cultures.

NASAL OPERATIONS.

Wexler[53] states that indications for surgical correction of the deviated nasal septum in children are nasal obstruction, chronic sinusitis, epistaxis and deformity of the nose after severe trauma. He emphasizes that only those portions of the nasal septum that are responsible for the presenting condition are removed.

Comment: Your reviewers admit that submucous resection of the nasal septum is necessary in some children. They feel, however, that very many children will have their septums operated upon if the indications of Wexler are followed, and point out that some of such interferences will be unnecessary and ultimately very unfortunate for the patient. Every child on whom submucous resection of the nasal septum is considered should be referred first of all to a rhinologist of long experience.

Gollom[54] discusses nasal fractures in both children and adults, their anatomical and pathological factors, their classifications and the management by the conventional closed method.

The anatomical problem in children is slightly different from that in adults. The nasal bones are separated by a suture line; the bones are softer, the cartilages thinner. With injury the bones may spread apart or the cartilages tear easily. Radiography may reveal soft tissue edema and nothing more. The septal cartilage may, with injury, buckle and spring from the vomerine groove, becoming dislocated into either the right or left nasal passage.

It is easy to overlook nasal fractures. In children particularly the rapid symmetrical swelling which may occur obscures the basic deformity. Palpation of a depressed bone is less certain. The intern in his hospital emergency department, or the family doctor, may be unaware that 40 to 50 per cent of nasal fractures will not show up on radiographs. A negative report gives the doctor who first sees the patient a false sense of reassurance. Four weeks later the patient or his family becomes convinced that a deformity really exists. By this time a relatively minor condition has changed into a major problem, due to bony union or scar tissue formation and contraction.

Reduction, where feasible, is best undertaken immediately after the accident. Where edema is considerable, reduction may be postponed for several days. Children require a general anesthetic. Manipulation and elevation of fragments will often accomplish disimpaction and reduction at the same time.

Following reduction of the fracture the nose is packed for four or five days and an external splint is applied and kept on for seven to ten days.

He also comments on the open rhinoplastic approach to more complicated nasal injuries.

Comment: It is the reviewer's opinion that an external splint prevents observation and that packing, in spite of antiseptic ointment, produces headache and perhaps infection. He prefers to do without either. He does favor putting restraining armlets on the child and at night preventing the child from rubbing its nose against the pillow.

TUMORS.

A case report of a thymic tumor of the pharynx is presented by Epstein and Loeb[55]. The presence of thymic tissue above the thyroid gland is rare, and the explanation is due to the fact that the thymus originates from the third and fourth branchial clefts.

The patient was a three-week-old infant in whom the mother noted the appearance of a mass protruding from the mouth when the baby regurgitated. It had the appearance of a second tongue, being about 1 cm in diameter. The mass projected from the mouth for a few minutes, then disappeared into the mouth. X-ray examination with barium showed the mass to be alternately swallowed and regurgitated. The base of the mass was seen to be in the medial portion of the left pyriform sinus. The mass was removed with a nasal snare after a ligature was tied around the pedicle. The pathological report showed a mass 3.5 cm. long and 1 cm. in diameter. Cross section showed the interior to be made up of cords of yellow tissue set in pink membrane. Sections showed a polypoid structure consisting of isolated groups of thymic tissue containing Hassall's corpuscles.

A case of teratoma of the tonsil in the newborn is reported by Baugh and O'Donoghue[56]. The tumor produced respiratory obstruction, and at birth the infant was in a state of asphyxia pallida, and remained so after large amounts of mucus were extracted. There was some improvement in color after the administration of oxygen and 7 per cent CO-2, but breathing remained difficult. Further examination at this time revealed a tumor protruding from the posterior surface of the left anterior pillar of the tonsil. The growth was obstructing the pharynx entirely. An emergency enucleation of the tonsil was carried out with a tonsil guillotine, and breathing became much easier. There was some trouble with hemorrhage which was finally controlled, but the baby died about 36 hours later.

The pathological report showed a tumor mass consisting of a tonsil and two polypi projecting from the external surface. The larger polypus measured 15x10 mm., and the smaller one 10x3 mm. The tonsil measured 20x10x15 mm., and on section showed bony, cartilaginous and other white tissues. Histological examination showed a disorderly mixture of cartilage, cancellous bone, plain and striated muscle, neuro-epithelium, and renal and adipose tissues, together with clumped masses of cells which included lymphocytes, plasma cells, eosinophiles and multinucleated giant cells. A diagnosis of teratoma was made.

The author believes that this is the first case reported of a teratoma arising in the tonsillar fossa.

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ANNUAL ASSEMBLY IN OTOLARYNGOLOGY.

The Department of Otolaryngology, University of Illinois College of Medicine, announces its Annual Assembly in Otolaryngology from October 1 through 7, 1956. The Assembly will consist of an intensive series of lectures and panels concerning advancements in otolaryngology, and evening sessions devoted to surgical anatomy of the head and neck, and histopathology of the ear, nose and throat.

Interested physicians should write direct to the Department of Otolaryngology, 1853 West Polk Street, Chicago 12, Ill.

AN EVALUATION OF THE PSYCHOGALVANIC SKIN-RESISTANCE TECHNIQUE IN AUDIOMETRY.*†

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Baltimore, Md.

Accurate determination of hearing loss in the very young child, and in certain adults, is a challenge to the otologist. Only with accurate determinations can our knowledge of the etiology of hearing damage be advanced or the proper plans be laid for the rehabilitation of these patients.

Hard-of-hearing children do not develop along the same lines as their chronological counterparts with normal hearing.[1] Without the natural means to communicate, learning becomes a very complicated and slow process. Such deviant development frequently obscures the clinical picture, even masking the basic handicap of hearing loss. Many of the children under the age of five years, seen in large clinics, are brought in with the admission diagnosis of "mental retardation", "aphasia", or "autism".

Children with partial hearing, especially those with residual hearing only for the low tones, suffer from failure to comprehend conversation or to develop speech; but at the same time it is evident to close observers that these children can hear. Speech being mere imitation of what one hears, a child with such a handicap cannot hope to produce much more than a series of "grunts". His communication being thus reduced to almost an animal level it is not surprising that many of them are stigmatized as mentally deficient and so denied the chance of early rehabilitation.

Adults suffering from various types of psychic trauma, or of brain damage, make up a smaller group of patients that present, with respect to testing hearing, a similar problem to

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that of the very young child. Subjective tests are unreliable. Even in those instances where malingering tests show the subjective test inaccurate, the problem of establishing accurate thresholds still remains.

Standard hearing tests used in diagnostic work in adults are inapplicable to the problems of the young, hearing-handicapped child and the adult with psychic trauma or brain damage, because they depend primarily on the voluntary cooperation of the examinee.

To obtain diagnostic information on the hearing status of deafened young children, four types of tests had been evolved by 1947: *1.* the use of various noise makers; *2.* speech audiometry; *3.* play audiometry; *4.* changes in electro-encephalograph records under the exhibition of a test tone.

The use of noise-making toys, which has been employed for many years,[2,3,4,5] reached a very refined state in the hands of the Ewings and Utley. They, separately, developed a set of calibrated noise makers which they used for testing; at times they substituted pure-tones during the test situation. Their accurate observations led to many successful determinations of hearing loss. Myklebust used a variation of this technique in his clinic. Such tests closely approach Play Audiometry. Their chief handicaps are: *1.* in many instances a response to vibration may be elicited as well as, or instead of, an auditory response; *2.* such tests are basically bilateral in character; *3.* it is seldom possible to make accurate determinations for thresholds of pure tones, and often impossible to distinguish where in the sound spectrum the greatest loss of hearing occurs.

Speech audiometry was used successfully by Keaster[6] and others[7 8] before 1947. By its use much information can be obtained about these children, whose speech development permits even limited communication. The difficulties encountered in using this technique result not only from its dependence upon cooperative communication, but also from the fact that it gives no information about the pure-tones involved or their individual thresholds.

Play audiometry reached its present excellence when Dix and Hallpike[9] developed the "Peep Show". Here a child is conditioned to press a button when he hears a pure-tone in order to be rewarded by the sight of an interesting picture. In expert hands and working with cooperative children this has proved an accurate and very satisfactory technique. Success here depends entirely upon the active cooperation of the examinee. The principal difficulties encountered have been *1.* with children under three years of age; *2.* with children suffering from psychic trauma; *3.* with those children with brain damage. Especially notable in this latter group is the child suffering from cerebral palsy.

Michels and Randt[10] in 1947 reported changes in the electroencephalograms of adult patients stimulated by pure-tones. Similar work was also reported by Marcus, Gibbs and Gibbs[11] in 1949 working with young children. The problems encountered in using this technique are: *1.* repetitions of the sound stimulus result in gradual disappearance of "response changes" in the tracings; *2.* the stimulating tone has to be delivered at a considerably greater intensity than the actual threshold in order to obtain a response; *3.* some tones in the sound spectrum produce better responses than others.

In an effort to obtain information on the hearing of adults and children who could not be satisfactorily tested by the foregoing techniques, Psychogalvanic skin-resistance (PGSR) audiometry was first attempted at the Johns Hopkins Hospital in 1947. This method of testing is based on the Pavlovian conditioned reflex.[12] Such a reflex had been demonstrated by Pavlov[13] before 1912. He repeatedly fed dogs soon after sounding a tone. After sufficient repetition of this sequence, the dogs would salivate at the sound of the warning tone in anticipation of the feeding. This was a sympathetic nervous system response to an auditory stimulus.

The changes in skin-resistance to the passage of a small electric current following sensory, or emotional stimuli, were first demonstrated in 1888 by Feré.[14] In 1928 Richter[15] developed a technique whereby he was able to record graphically the qualitative changes in skin-resistance following stimulation of the sympathetic nervous system by an electric shock. For this determination he used a delicate Wheatstone bridge in circuit with a direct current millivolt amplifier, coupled with a graphic

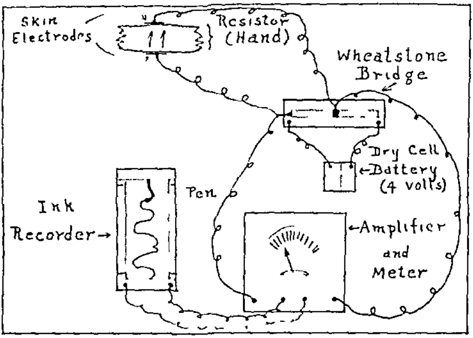


Fig 1 Schema of the circuit used to record changes in skin resistance

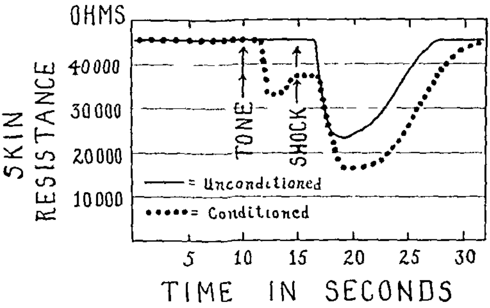


Fig 2 Diagrammatic graph of the changes in skin resistance in a person before and after conditioning to a tonal stimulus

recorder. The shock device was a Harvard inductorium with which the intensity of the shock could be varied. Richter's technique consisted of adjusting the Wheatstone bridge so that the least current possible was passed through the skin, usually of the order of 2-20 microamperes. The patient was then mildly shocked, which resulted in a sympathetic stimulus. This caused increased activity of the sweat glands and a lowering of skin-

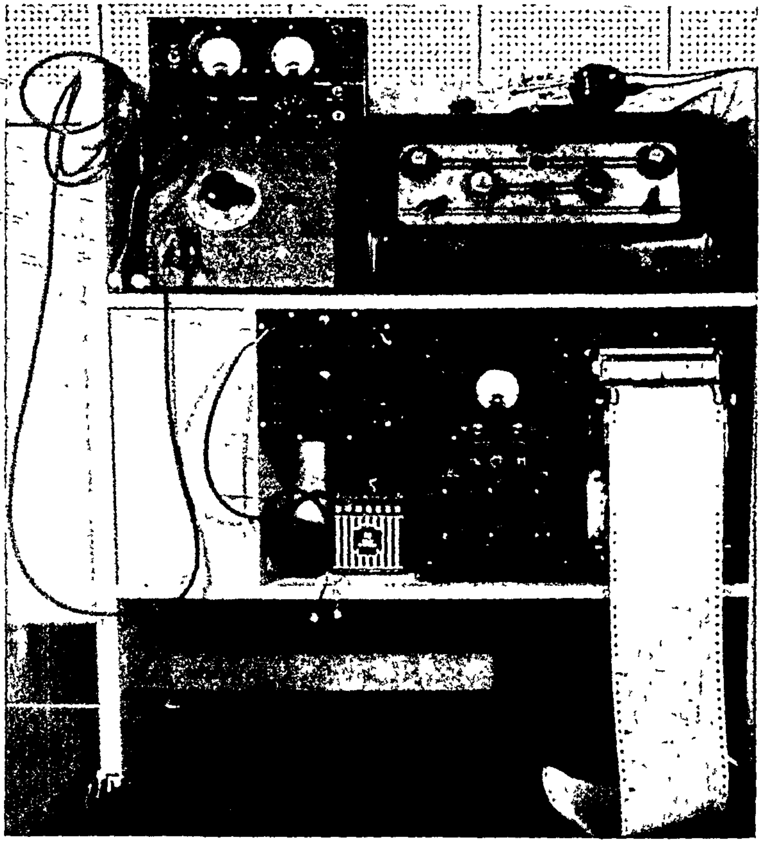


Fig. 3. The apparatus used in PGSR audiometry. The shock device and the audiometer are above the Wheatstone bridge, the amplifier and the inkwriter.

resistance. Such skin-resistance changes were amplified and recorded.

The PGSR technique of testing hearing acuity employs Richter's equipment,[16] adding to it a standard audiometer. A pure-tone from the audiometer, through the headphone, is used as a warning signal of the coming shock, which follows in approximately five seconds. After the warning tone-shock sequence is repeated for several minutes the patient develops a sweating response after the sound in anticipation of the coming shock. When this occurs, the patient can be said to have developed a "conditioned reflex". Thereafter, as long as the patient hears a warning tone there is a drop in skin-resistance before the shock. When the tone is not heard the patient receives no warning of the coming shock, and there is no premature sweating response (see Figs. 1 and 2).

Fig. 3 shows the skin-resistance audiometer. Fig. 4 shows the test situation. The shock electrodes will be noted on the leg and the skin-resistance electrodes can be seen on the finger tips. The shielded shock device and standard audiometer are placed above the Wheatstone bridge, the amplifier and recorder.

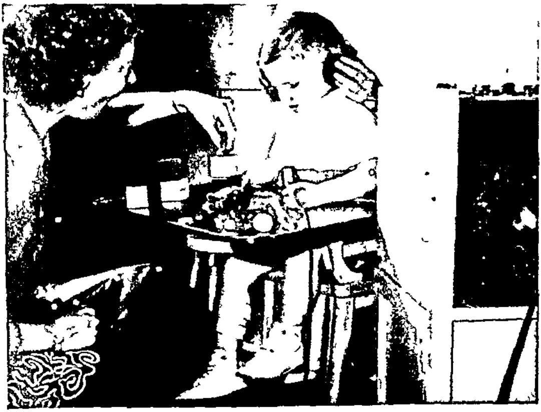


Fig 4 The test situation The shock electrodes are on the right calf, the skin-resistance electrodes are on the left hand

Fig. 5 represents an actual tracing of a patient who has been conditioned and whose threshold is being determined for the frequency of 1024 cps. It can be seen how the premature response occurs before the shock, and how, when the tone is dropped below threshold, the skin-resistance changes only after the shock.

Long experience with this method has shown that once an

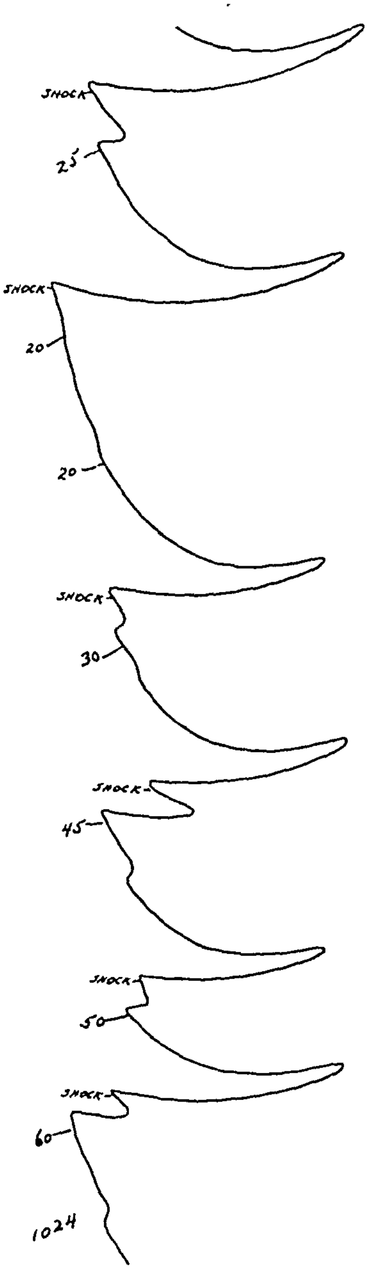


Fig. 5. An actual tracing obtained during PGSR audiometry, illustrating the method of determining the threshold value for a pure tone in a subject in whom a conditioned reflex has been established.

individual is conditioned for one pure-tone he is conditioned for tones in all octaves which may be used in the test in either ear.[17] Such conditioning lasts approximately 30 minutes after completion of the testing. Unless the shock is kept at a noxious level there is a general tendency for conditioning to fade when the test is carried on over a prolonged period of time. This may require increasing the intensity of the shock several times during a test. A masking sound may be used in the opposite ear without invalidating the test.

By using this method thresholds can be plotted on a normal audiogram form just as in subjective audiometry. The period of time necessary for conditioning varies with each individual. Usually it can be accomplished within ten minutes. The length of time to obtain an audiogram usually ranges from 20 minutes to an hour. One hour should be the limit for such testing; because after that period the subject becomes extremely tired, and the complicating factors of sleep and exhaustion enter the picture.

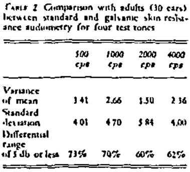


Table 2 Comparison with adults (30 ears) between standard and galvanic skin resistance audiometry for four test tones

	500 cps	1000 cps	2000 cps	4000 cps
Variance of mean	3.41	2.66	3.50	2.36
Standard deviation	4.01	4.70	3.84	4.00
Differential range of 5 db or less	73%	70%	60%	62%

Fig. 6.

How Accurate Are Such Audiograms? For the first three months this method was employed it was used only on adults, and whenever a PGSR audiogram was made it was followed by a subjective audiogram. Individuals chosen for such studies included normal to extremely hard-of-hearing patients. Records on 30 ears of 20 individuals with impaired hearing revealed an average difference of less than five dbs between subjective and objective audiometry throughout the sound spectrum (see Fig. 6).

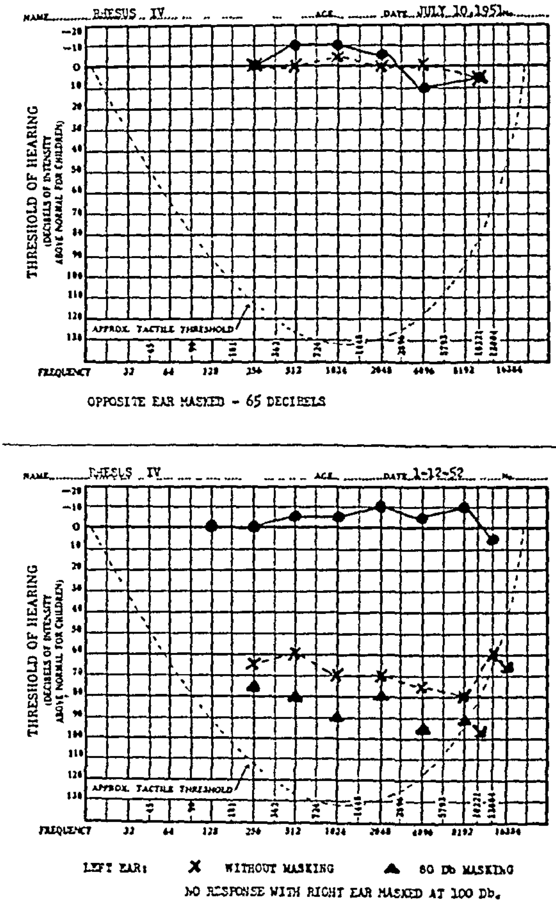


Fig. 7. PGSR audiograms obtained from a rhesus monkey before and five months after a complete section of the left auditory nerve.

What Is Being Tested by the PGSR Method? The end-organ for this reflex lies within the ear, and the responses obtained are not the reaction to some tactile stimulus. This fact has been demonstrated by sectioning one auditory nerve of a previously tested Rhesus monkey, allowing the monkey to make a complete physical recovery and retesting his hearing. The unoperated ear gives a normal audiogram, while the ear in which the auditory nerve has been sectioned, when the other ear is properly masked, gives no response to auditory stimuli (see Fig. 7).

The localization of the region in the brain for the mediation of this reflex is, as yet, unestablished. Pavlov[13] in 1912 theorized that the site of such a conditioned reflex must lie within the cerebrum. It is now thought that there may be several levels in the brain at which this reflex may be mediated. For this type-testing the lowest level of communication between the auditory pathways and the sympathetic nervous system, would seem to be of great importance. Injury to auditory pathways or auditory projections above the lowest level of crossover, might not reflect itself in such a conditioned reflex audiogram.

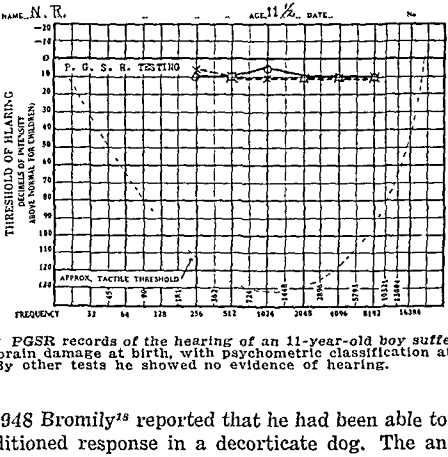
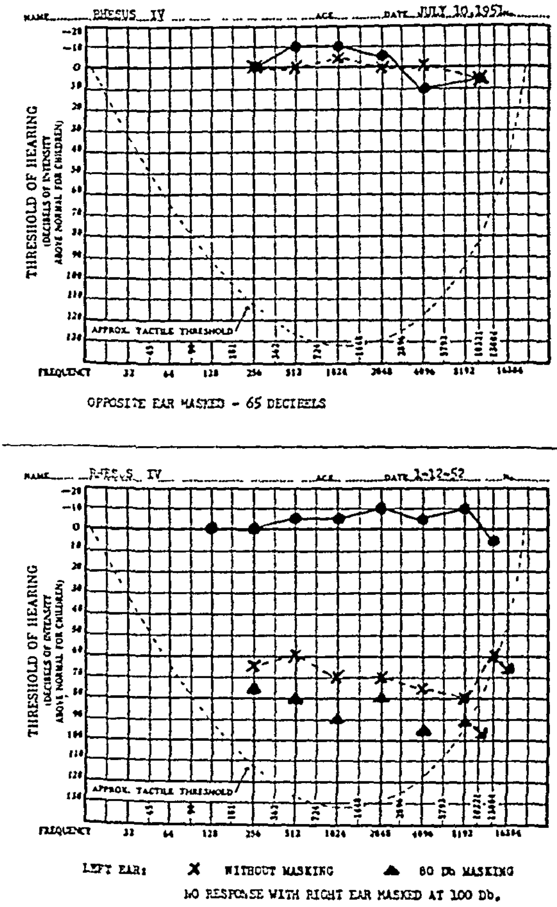


Fig. 8 PGSR records of the hearing of an 11-year-old boy suffering from severe brain damage at birth, with psychometric classification at the idiot level. By other tests he showed no evidence of hearing.

In 1948 Bromily[18] reported that he had been able to develop a conditioned response in a decorticate dog. The animal retained the power of discrimination between light and sound. Wang and Nager[19] have recently conditioned a decorticate cat to two types of auditory stimuli (bell and buzzer) by the PGSR method. In 1954 Wang, working in Richter's laboratory, performed some as yet unpublished experiments, in which he abolished the shock-sweat reflex in cats by transection of the brain from the intercollicular level to the rostral border of the pons. Transection above this level did not abolish the reflex. It would seem then, that the auditory-sympathetic reflex



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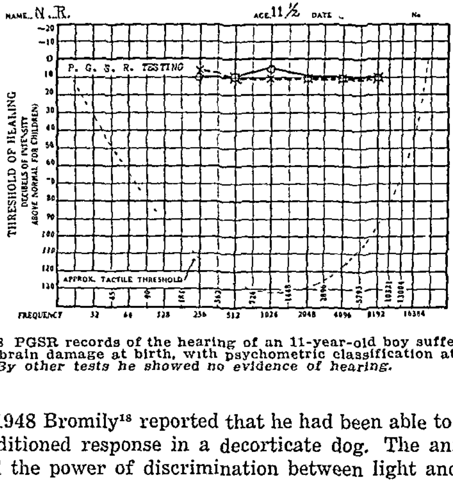

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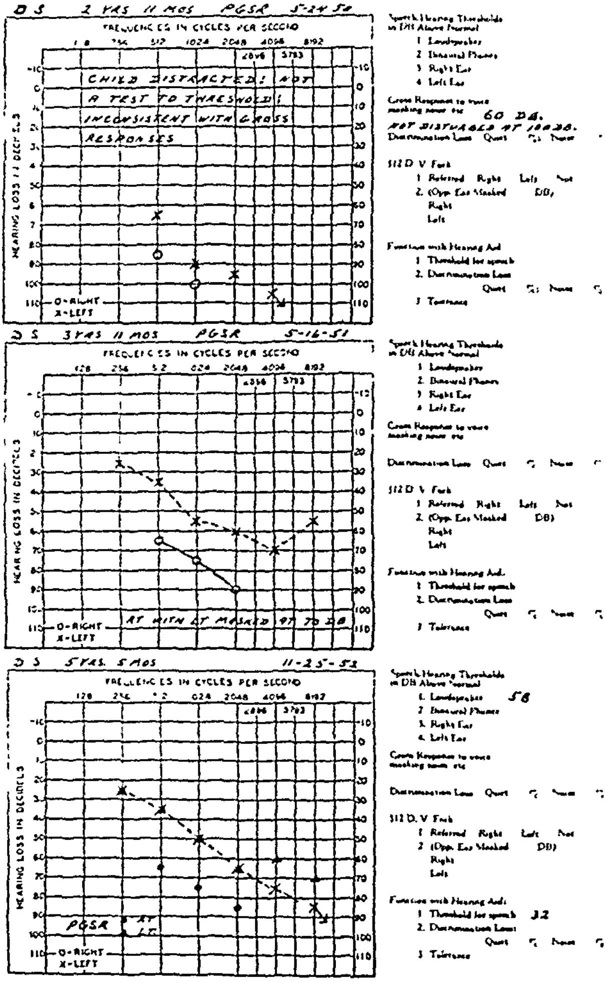


Fig. 9. Example of the "stabilization" of hearing levels of a young child after repeated tests by PGSR audiometry. (2nd audiogram). Comparison of PGSR values for left ear with standard audiometry 18 months later. (3rd audiogram).

must lie somewhere below the cortex and above the level of the inferior colliculi and the rostral border of the pons.

That such an audiogram represents a test of the efficiency of the auditory end-organ rather than any comprehension of sound is further demonstrated by the little boy, age 11 years (see Fig. 8), suffering from severe brain damage as the result of birth injury, severe prolonged anoxia and multiple post-partum convulsive seizures. He did not walk until he was three-and-a-half years old, and even at 11 he had a wide unsteady gait. He was not toilet trained, and would feed himself only occasionally, using his hands instead of a spoon. He had marked disturbances in his electroencephalogram. He was at idiot level by psychometric evaluation. He had developed speech. He occasionally would turn his head toward some sound. He gave no satisfactory response to standard hear- tests, *yet by PGSR audiometry his hearing was normal.*

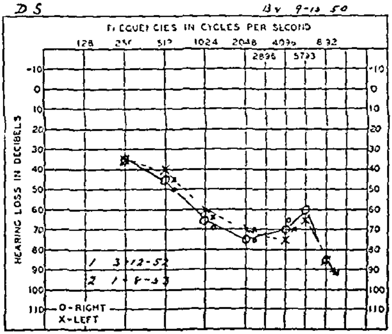


Fig 10. The test-retest PGSR audiograms of a child who was first tested at 19 months of age, and again at 28 months

He had no hearing in the broadest sense of the word, but his end-organ function was unimpaired. His reception was unimpaired, but his perception was destroyed.[20]

It is felt that when testing children the initial audiogram represents hearing at least as good as the thresholds obtained. We do not consider this the true threshold, however, until we have obtained at least two audiograms on separate occasions that average within five dbs of each other. To obtain such determinations has occasionally required as many as three or four test periods (see Fig. 9). Each succeeding test in these children shows an improvement.

Such improvement subsequent to initial testing has also been noted by Barr[21] in both play audiometry and psychogalvanic audiometry. The Ewings[22] in a follow-up study of the hearing levels of a group of children whose thresholds had been established by their "Toy Tests" found that no child tested worse than his first level. A number of the children were found to have better thresholds in the follow-up tests. This variation is seen more frequently in younger children under three-and-one-half or four years. It is also more likely to occur in the presence of some brain injury. Once the threshold has been established the test-retest picture has proven satisfactory (see Figs. 10 and 11).

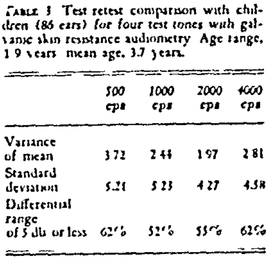


TABLE 3 Test retest comparison with children (86 ears) for four test tones with galvanic skin resistance audiometry Age range, 1-9 years mean age, 3.7 years.

	500 cps	*1000 cps*	*2000 cps*	*4000 cps*
Variance of mean	3.72	2.44	1.97	2.81
Standard deviation	5.21	5.23	4.27	4.58
Differential range of 5 db or less	62%	52%	55%	62%

Fig. 11. Statistical evaluation of test-retest values on 86 ears of children ranging from one to nine years.

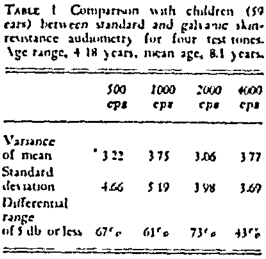


TABLE 1 Comparison with children (59 ears) between standard and galvanic skin-resistance audiometry for four test tones. Age range, 4-18 years, mean age, 8.1 years.

	500 cps	*1000 cps*	*2000 cps*	*4000 cps*
Variance of mean	3.22	3.75	3.06	3.77
Standard deviation	4.66	5.19	3.98	3.69
Differential range of 5 db or less	67%	61%	73%	43%

Fig. 12.

In very young children it is impossible to make objective-subjective cross checks on audiograms. Standard pure-tone audiometry is rarely reliable under six years of age. A series of 59 ears on 39 children ranging from four to 18 years, and averaging eight years, were studied by Hardy and Bordley[23] by PGSR and standard audiometry. Deviation was less than 5 db in from 61 to 73 per cent of the tests at 500 cps., 1000 cps. and 2000 cps. The average of deviations shows that the threshold for PGSR is slightly lower than that obtained by standard audiometry (see Fig. 12). These data are slightly at variance with the reports of Barr,[21] Faure,[24] and Maspitiol[25] in similar groups of children.

Barr states that standard audiometry is about 5 db below thresholds obtained by PGSR. Faure reports in his series that true thresholds lie about 15 db below PGSR thresholds in examinees between two-and-one-half and four years, and about 10 db below in the four to seven year group. Maspitiol, Gougerot and Korine observed a 15 to 20 db discrepancy against PGSR thresholds in the children they studied between four and eight years of age. Barr in his recent monograph states that thresholds established by play audiometry and PGSR audiometry, once they have become stabilized, showed substantial agreement. Less than 15 per cent of the audiograms showed more than 5 db difference when checked by both methods, in a group of 68 children from two to six years old.

What Is the Earliest Age at Which Such Audiograms Can Be Attempted? Here again it depends on the individual. In general by the age of 18 months, testing a child without severe brain damage, should be relatively free from difficulty. Younger than this the child's skin-resistance seems to increase, and the sympathetic response becomes slower. In many instances we have encountered in the child of six months, a skin-resistance so great that our Wheatstone bridge, which is capable of handling up to 300,000 ohms resistance, cannot overcome it. We have successfully tested many children less than one year old, but failure increases greatly under 18 months of age. Such increased difficulty was also noted by Barr, who stated that he could not anticipate success in establishing thresholds by PGSR audiometry in more than 50 per cent of patients under two-and-one-half years of age.

LaBenz,[26] in using this technique experimentally on newborn infants, has found that the sympathetic response to shock in them is very greatly slowed down, resulting in a much greater time lag between stimulus and response. He also found the most active sweat glands to be on the head, instead of on the hands and feet. Altogether he studied 41 children averaging 134 hours old and was able to elicit responses to sound stimulation in 32 per cent. No attempt has been made to interpret these responses as thresholds, or to consider them as conscious levels of hearing.

What Difficulties and Failures Are Encountered in Using PGSR Audiometry to Test Children? As with almost any type of technique to test hearing, failures have been encountered when using this method. Difficulties have been encountered in conditioning some children. In a high percentage of cases difficult to condition, follow-up studies have subsequently shown evidence of brain injury. Difficulty has been encountered in evaluating records on athetoids and spastic children because of the many false positives due to extraneous bursts of sympathetic stimuli in these children.

One of the greatest problems is for the test personnel to maintain an "objective attitude". Some of the worst mistakes have resulted from the tester distorting the interpretation of questionable tracings to fit his preconceived ideas. Goldstein[27] has also emphasized this weakness. When records are not clear they should be discarded. The incidence of such failures is certainly not over four to five per cent, which we feel is not sufficient to invalidate the method.

The percentage of failures has steadily decreased as we have improved our techniques in handling the child in the test situation. This is extremely important and can be done by a well-trained team, which through experience can keep the child relatively quiet and fairly well occupied during the tests.[28] Many children have to be retired from the test situation and allowed to take a nap, or even get a night's rest, when they become severely disturbed; but with a well-trained team that is willing to devote unlimited time to a child, success is the rule rather than the exception. Few children show serious fear disturbances when the test situation is properly handled. Barr in his series found that less than 5 per cent showed disturbing signs of fear.

PGSR audiometry has been employed in the hearing studies of over 1600 adults. Many of these patients have been examined to obtain validating studies on the PGSR method, but more than 400 have been tested by this technique because there was reason to question the accuracy of the standard subjective audiometric findings. As a group adults are far easier to condition and test than children. It is thought that such ease of testing results from the fact that the method acts as a "lie detector" as well as a conditioner. The problem of stabilizing the threshold of response to a tone does not seem to present itself in adults as in young children.

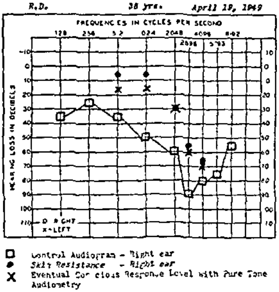


Fig 13. Standard and PGSR audiograms of the right ear of a 36-year-old woman with a "psychogenic overlay." Note that a repeat subjective audiogram, done after the PGSR study, showed marked improvement

The first audiogram is usually correct. Failure to establish satisfactory hearing levels has occurred in less than 2 per cent of the patients examined. Two complete failures occurred in testing two individuals suffering from a deep catatonic state. It has been used successfully in four general groups of patients: *1.* those suffering from some "psychogenic overlay" (see Fig. 13) ; *2.* malingerers (see Fig. 14) ; *3.* those adults with severe hearing loss who have grown up in a deaf atmosphere such as a "signing" school situation, and who as a result *have never developed any ability to listen* (see Fig. 15) ; *4.* persons with certain types of brain lesions.

This latter category is of particular interest, as it offers a different diagnostic use for PGSR. It is comprised of older

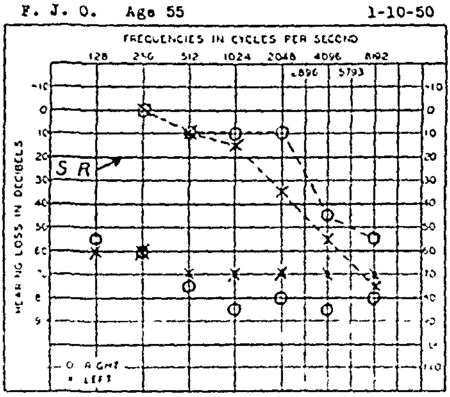
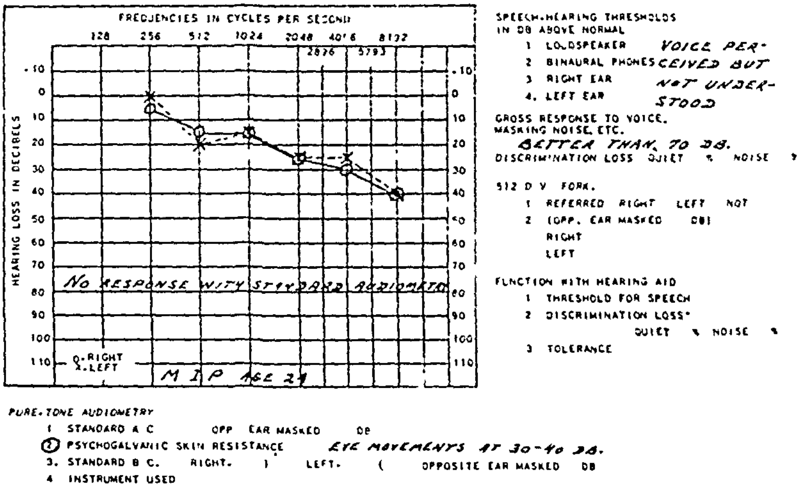


Fig. 14. Standard and PGSR (SR) audiograms of a 55-year-old man seeking compensation for an old head injury.



SPEECH-HEARING THRESHOLDS
IN DB ABOVE NORMAL
1 LOUDSPEAKER VOICE PER-
2 BINAURAL PHONES CEIVED BUT
3 RIGHT EAR NOT UNDER-
4. LEFT EAR STOOD

GROSS RESPONSE TO VOICE,
MASKING NOISE, ETC.
BETTER THAN, 70 DB.
DISCRIMINATION LOSS QUIET % NOISE %

512 D V FORK.
1 REFERRED RIGHT LEFT NOT
2 (OPP. EAR MASKED DB)
RIGHT
LEFT

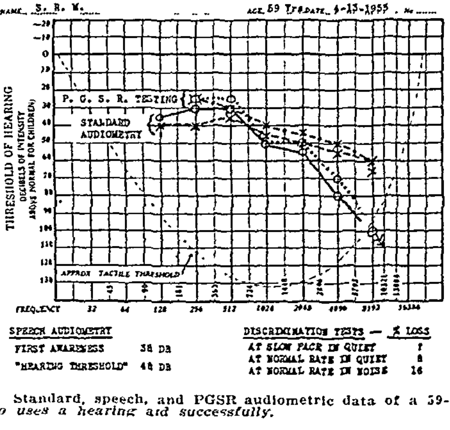
FUNCTION WITH HEARING AID
1 THRESHOLD FOR SPEECH
2 DISCRIMINATION LOSS:
QUIET % NOISE %
3 TOLERANCE

PURE-TONE AUDIOMETRY
1 STANDARD A C OPP EAR MASKED DB
② PSYCHOGALVANIC SKIN RESISTANCE EYE MOVEMENTS AT 30-40 DB.
3. STANDARD B C. RIGHT.) LEFT. (OPPOSITE EAR MASKED DB
4 INSTRUMENT USED

Fig. 15. PGSR audiogram of a 24-year-old woman, raised from infancy as a deaf child, educated in a deaf school environment. She had good speech, but gave no responses to standard audiometry.

adults. By subjective hearing studies they appear to be satisfactory candidates to wear hearing aids, yet they have never been successful in their efforts to use them. Such individuals are familiar to every Hearing Center and offer a very baffling problem to otologist and audiologist alike.[29]

A group of 20 of these patients have been under study by Bordley and Haskins[20] for the past two years. They have been tested repeatedly by standard subjective pure-tone audiometry, *PGSR and speech audiometry; they have all been individuals* distinguished by success in their chosen fields of work. In each case the hearing difficulty has arisen in the preceding two to four years. They feel that they can hear quite well, but can no longer discriminate words. The problem is greatly accentu'ed in group communication, or whenever the listening sit-tion becomes complicated.



SPEECH AUDIOMETRY
FIRST AWARENESS 38 DB
"HEARING THRESHOLD" 48 DB

DISCRIMINATION TESTS — % LOSS
AT SLOW PACE IN QUIET 7
AT NORMAL RATE IN QUIET 8
AT NORMAL RATE IN NOISE 16

Fig. 16 Standard, speech, and PGSR audiometric data of a 59-year-old *man who uses a hearing aid successfully.*

Their hearing tests have the following in common:

1. The PGSR audiogram indicates significantly better hearing than does the standard subjective audiogram.

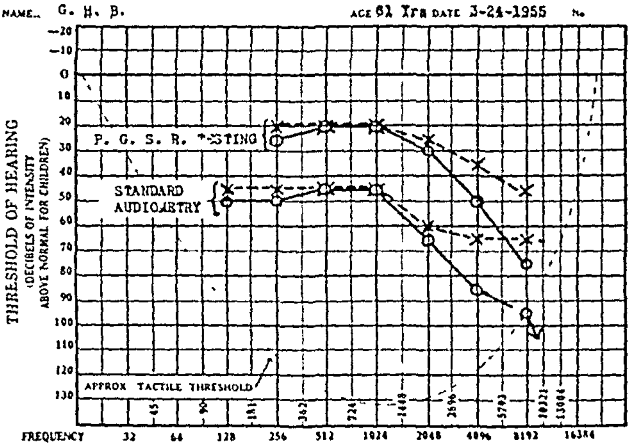
2. Thresholds in decibels by speech audiometry for awareness of sound and for understandability of speech are much further separated than in those persons using *amplification* successfully.

3. Ability to understand single simple words when spoken slowly is good. As the speed of delivery is increased there is *an abnormal breakdown in the understanding.*

4. Discrimination breaks down abnormally in noise.

A comparison of the hearing test records of a successful hearing aid user with one of the study group will highlight the significant difference in the responses (see Figs. 16 and 17).

The consistent difference between PGSR audiometry and standard audiometry in all of these patients is significant. PGSR audiometry has been shown to be dependent upon the intactness of the auditory end-organ and auditory pathways at least up to the level of the colliculi, but does not seem to require an undamaged cerebral cortex. Standard pure-tone audiometry, depending upon subjective responses, would seem to require not only an intact auditory end-organ and nerve but also undamaged higher pathways, and cortical projections.



SPEECH AUDIOMETRY	
FIRST AWARENESS	26 DB
"HEARING THRESHOLD"	46 DB

DISCRIMINATION TESTS —	% LOSS
AT SLOW PACE IN QUIET	4
AT NORMAL RATE IN QUIET	38
AT NORMAL RATE IN NOISE	46

Fig 17. Standard, speech, and PGSR audiometric data of a 61-year-old man, who has suffered repeated failures in attempts to use a hearing aid.

The difference then, between the two types of tests in this group of individuals who have poor discrimination, a poor standard audiogram, and a good PGSR audiogram, would point to some damage to the higher auditory pathways. Such damage does not interfere with sound reception but rather to its perception. Such damage would seem to be cerebral, probably in the cerebral cortex.

Such a theory is supported by the work of several animal experimenters. Babkin[30] working under Pavlov in 1911, reported tests on a dog which survived three years after removal of the posterior portion of the cerebral hemispheres. Before operation the dog had been conditioned to sequences of sounds, but after operation he could never be taught to discriminate between tones. In 1912 Pavlov[11] observed "From the standpoint of the conditioned reflexes the cerebral hemispheres appear as a complex of analyzers whose functions are: to decompose the intricacy of the outer and inner worlds into their separate elements and components; and further, to connect all of these with the manifold activity of the organism." Meyer and Woolsey[31] in 1952 reported that after bilateral ablation of the auditory cortex some animals could be conditioned to respond to a sound but could no longer discriminate between frequencies. Diamond and Neff[32] in 1953 reported that cats after bilateral ablation of the auditory cortex lost the ability to discriminate between tones. All these experiments indicate that damage to the auditory cortex results in damage to auditory perception, rather than to auditory reception.

The theory that those individuals with poor discrimination, poor subjective audiograms, and good PGSR audiograms are suffering from damage to the auditory cortex rather than the auditory end-organ and nerve, will also explain a group of cases that have previously been reported in the literature. In 1934 Crowe, Guild and Polvogt[33] reported a group of ears which showed no pathology in the end-organ or auditory nerve, although standard pure tone audiometry had revealed a nerve type hearing loss. In 1937 Fieandt and Saxen[34] reported similar findings. In 1955 Schuknecht[35] added to this growing list and suggested, as had Crowe, that such hearing losses might be the result of central auditory pathway lesions. He went further in his speculation and theorized that degeneration of second, third and fourth order neurons might be significant in old-age deafness.

PGSR audiograms on these individuals might well have been much better than the standard audiograms, thus pointing to a possible cortical damage.

DISCUSSION.

PGSR audiometry would seem to measure only the efficiency of the auditory end-organ and the subcortical auditory pathways, while standard audiometry measures the efficiency of the hearing mechanism as a whole from the external ear through the auditory cortex. It evaluates the reception rather than the perception of sounds. This difference should always be borne in mind in the evaluation and interpretation of the findings of PGSR studies. The more limited function of PGSR audiometry has great advantages over the standard methods of pure-tone audiometry, speech audiometry, and play audiometry in certain instances.

Children under six years of age cooperate so poorly[36] that under even good circumstances standard audiometry is so unreliable as to be rejected by a majority of those most versed in evaluating their hearing. This would reduce pure-tone audiometry to two methods, play audiometry, and PGSR audiometry. Play audiometry, where there is good cooperation and enough communication, offers certain definite advantages; it tests the whole hearing mechanism. In situations where hearing determinations are necessary in the brain-injured child who cannot cooperate, such as in cerebral palsy, in the child whose hearing loss has delayed his ability to communicate, or in the child under three years of age, PGSR audiometry would offer a logical solution.

It seems to be true in all types of hearing determinations in young children, that retest is required to establish a stabilized threshold. In some cases the exact measurement of hearing cannot be established for a matter of months or longer; nevertheless, because a child's ability to learn the communicative skills seems to be optimum in the period between one and four years, the importance of the early hearing evaluation cannot be too strongly stressed. In all hearing evaluation in young children one should keep in mind the difference between apparent and potential thresholds.

Some children whose hearing is evaluated by PGSR audiometry and found to be satisfactory may eventually manifest evidence of brain damage. This danger is always inherent in a test that does not evaluate the hearing mechanism as a whole; but such a test has the inestimable value of establishing whether the deviant development of a child is the result of some peripheral lesion or a more serious and probably more pervasive central lesion.

The chief difficulties encountered in testing the hearing of children by PGSR are found: *1.* in establishing conditioning in the presence of certain brain injuries; *2.* in the building of a team capable of handling the test situation; *3.* in the unbiased interpretation of the skin-resistance tracings.

The need of PGSR audiometry in adults is more limited, although its technical difficulties are fewer than those encountered in children. The categories of psychoses and malingering have been under investigation by the PGSR technique in several University clinics and in the audiology section of the Veterans Administration, and the findings have warranted its continued use. Combining standard and speech audiometry with PGSR audiometry would seem to offer a method for study of damage to the auditory pathways in the cerebrum and the auditory cortex.

CONCLUSION.

It is our considered opinion that PGSR audiometry offers a valuable adjunct to other methods for hearing evaluation. It is particularly useful in studying the hearing of young children and certain adults. It measures reception rather than perception of sound. For objective determination of thresholds of hearing for pure-tones, it offers a clinically practical approach. It can be employed in the animal experimental laboratory. It does not necessarily evaluate hearing at the cortical level. Its accuracy is greatly enhanced when it is undertaken by highly trained personnel. It is not yet free from failure, and probably never will be.

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SIXTH INTERNATIONAL CONGRESS ON OTOLARYNGOLOGY.

The deadline for submitting titles of papers and films for presentation at the Sixth International Congress of Otolaryngology is Oct. 1, 1956. Requests for places on the scientific program must be received before that date together with an 800-word abstract of the paper. Applications and abstracts should be sent air mail to the General Secretary, 700 N. Michigan Ave., Chicago 11, Ill., U. S. A.

SIGNIFICANCE OF OTOLOGIC FINDINGS IN CEREBELLOPONTINE MENINGIOMA.*

HENRY A. BROWN, M.D.,
and
J. GRAFTON LOVE, M.D.,
Rochester, Minn.

REVIEW OF THE LITERATURE.

In 1922, Cushing[1] proposed the term "meningioma" to designate a group of tumors originating from the meninges of the brain and spinal cord. Meningiomas are slow-growing tumors originating from the "cap cells" (meningiocytes), which tend to collect in clusters around the tips of arachnoid villi and are derived from the neural crest.[2] These tumors rarely invade cerebral tissue but push it aside. They may become extremely large and may be present for many years before producing clinical evidence of intracranial disease.[2] Kernohan and Sayre[2] considered that 36 of a series of 370 intracranial meningiomas were malignant. They stated that these tumors practically never give rise to metastasis either within or outside the cerebrospinal axis. Their criteria for malignancy were purely on histologic grounds. Meningiomas may come in contact with the skull and produce changes that lead the roentgenologist to suspect the identity of the disease.

Meningiomas make up a considerable percentage of all intracranial tumors. In the experience of Shelden and Adson,[3] this figure was 15 to 25 per cent, whereas in that of Horrax and Strain[4] it was 14.6 per cent and in that of Cushing[5] it was 13.5 per cent.

Kernohan and Sayre emphasized the several loci of predilection of meningiomas. Of their total of 794 cranial meningiomas, 184 were parasagittal, 73 were attached to the sphenoidal ridge, 26 were found in the basofrontal region or the olfactory groove, 61 were around the sella turcica, and 55 were found

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*Submitted as Candidate's Thesis to American Laryngological, Rhinological and Otological Society, Inc., 1956.

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at the cerebellopontine angle. In a similar study of 313 such tumors, Cushing and Eisenhardt[6] stated that 65 were parasagittal, 56 were over the free convexity, 53 came from the sphenoidal ridge, 29 came from the olfactory groove, 28 were suprasellar, 23 were in the posterior fossa, and lesser numbers originated elsewhere in the meninges.

Hamby[7] described two patients who had trigeminal neuralgia due to contralateral meningiomas of the posterior fossa.

Russell and Bucy[8] listed meningiomas of the posterior fossa as being a tenth as frequent as supratentorial meningiomas and stated that one of 10 tumors of the posterior fossa was a meningioma; these authors reported 15 cases of meningioma of the posterior fossa, in five of which the tumor was situated at the cerebellopontine angle.

Woltman and Love[9] emphasized that meningiomas may produce extremely few recognizable signs and symptoms even after reaching a huge size.

Bager[10] found meningiomas at the cerebellopontine angle to be only a fifteenth as common as acoustic neuromas of the same region.

Cushing and Eisenhardt reviewed seven cases of meningioma of the cerebellar chambers simulating acoustic neuroma and concluded, even after knowing the diagnosis, that the clinical findings were still sufficiently typical to justify a diagnosis of acoustic neuroma.

Brown and Love[11] found some degree of impairment of labyrinthine function in all of 79 cases of acoustic neuroma in which adequate tests were done. In Ireland's[12] series of acoustic neuromas, 4.3 per cent were found to be associated with normally functioning equilibrial labyrinths.

Dix and associates,[13] as well as Eby and Williams,[14] reported that recruitment of loudness was present in cochlear lesions and was absent in injuries to the trunk of the VIIIth nerve brought about by tumors at the cerebellopontine angle.

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In studying a group of cases, the reviewer invariably regrets that in some instances the records are not more nearly complete, this is particularly true in neurologic patients, many of whom are too seriously ill to give complete co-operation The hearing and caloric tests in this series were carried out by several persons, and the methods used were not always the same If the data obtained in a given instance did not appear reasonably accurate, the case was not included in the study

Table 1

SYMPTOMS OF DYSFUNCTION OF THE VIIITH NERVE IN 55 CASES OF MENINGIOMA AT THE CEREBELLOPONTINE ANGLE

SYMPTOMS	Number	Per cent (of 55)
Loss of hearing	49	89
Total or severe loss of hearing on side of lesion	33	60
Moderate loss of hearing on side of lesion	10	18
Slight loss of hearing on side of lesion	6	11
Bilateral loss of hearing	15	27
Normal hearing	6	11
Loss of hearing as first symptom	30	55
Patients tested for recruitment	2	4
No recruitment present	2	4
Unilateral tinnitus	24	44
Bilateral tinnitus	5	9
Tinnitus as first symptom	5	9
Dizziness	22	40
Vertigo	6	11

Table 1 lists the symptoms of decreased function of the VIIIth cranial nerve in this series For emphasis, it appears pertinent to dwell briefly on some of the more important findings Deafness was present in 49 of these 55 cases of cerebellopontine meningioma (89 per cent) In 33 instances (60 per cent), the loss of hearing was severe or total on the side of the lesion, in 16 instances (29 per cent), the ipsilateral decrease in acuity of hearing was slight to moderate, and in six (11 per cent) of the cases, the hearing was normal

PRESENT STUDY.

This study represents an analysis of data in 55 cases of cerebellopontine meningioma in which operations were performed and the diagnoses confirmed microscopically. Seventeen patients were male and 38 were female. At the time of examination, the youngest patient was 12 years of age and the oldest 65. Three patients were less than 30 years of age and four were more than 60. Forty patients were between 40 and 60 years of age. No attempt was made to list the complete neurologic findings, the purpose being to examine the data from an otologist's viewpoint, to attempt to ascertain any signs or symptoms that might be helpful in the diagnosis of cerebellopontine meningioma, and to list findings in addition to those resulting from injury to the VIIIth cranial nerve that could be elicited with the equipment at hand and that, when found, suggested the presence of a lesion at the cerebellopontine angle.

This study is concerned primarily with meningiomas at the cerebellopontine angle; however, the difficulty of the differentiation of meningiomas from acoustic neuromas was made so apparent by a general review of tumors at the cerebellopontine angle that it appeared worth while to compare a series of these two types of tumors and list any findings that might aid in the differential diagnosis. Cysts, cholesteatomas, vascular anomalies, chemodectomas, neuromas of the Vth nerve and inflammatory lesions may produce similar findings, but only meningiomas and acoustic neuromas will be considered in this study.

SIGNS AND SYMPTOMS OF DECREASED FUNCTION OF THE VIIITH CRANIAL NERVE.

In the majority of instances, meningiomas at the cerebellopontine angle produce symptoms and signs readily recognized as due to a lesion in this region, even though the exact nature of the disease is not a certainty.

Evaluation of audiograms and the results of caloric tests, along with assessment of the proper significance of tinnitus and disturbed equilibrium, may aid in placing a given tumor at the cerebellopontine angle and occasionally offer evidence that the lesion is a meningioma.

In studying a group of cases, the reviewer invariably regrets that in some instances the records are not more nearly complete; this is particularly true in neurologic patients, many of whom are too seriously ill to give complete co-operation. The hearing and caloric tests in this series were carried out by several persons, and the methods used were not always the same. If the data obtained in a given instance did not appear reasonably accurate, the case was not included in the study.

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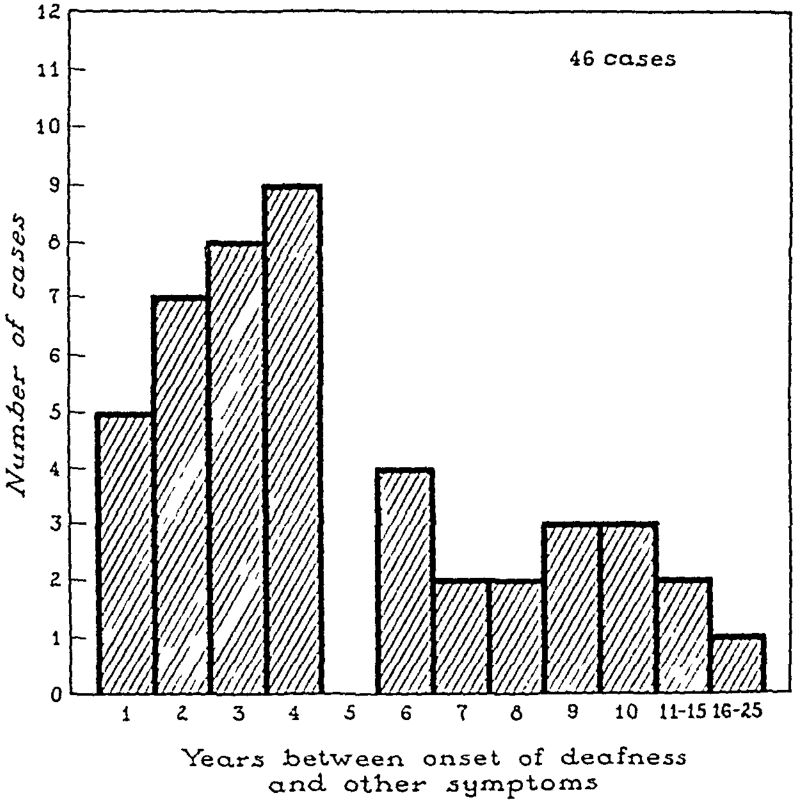

Fig. 1.

Frequency distribution of 16 cases of meningioma at the cerebellopontine angle by interval between onset of deafness and the appearance of other symptoms.

Deafness or tinnitus or both were the initial symptoms in 35 (64 per cent) of these 55 cases of cerebellopontine meningioma. Tinnitus was found in 29 (53 per cent) of the cases; it was bilateral in five instances. There was nothing diagnostic about the tinnitus, but it was frequently unusual in being intermittent. In one instance, it was present for a number of years and then disappeared. A few patients noticed tinnitus only with flare-up of other symptoms, such as headache.

Recruitment was absent in the two patients who were tested for recruitment in the deafened ear over the normal ear.

The length of time between the onset of deafness and the appearance of other symptoms that ultimately brought about the diagnosis of meningioma at the cerebellopontine angle is shown in the accompanying figure. Of the 46 patients from whom reasonably accurate histories could be obtained, 29 noticed symptoms other than deafness within four years. Symptoms of VIIIth-nerve dysfunction alone were present for more than 10 years in three patients, one of whom noticed deafness for 25 years before symptoms suggesting intracranial disease appeared.

Table 2.

RESULTS OF CALORIC TESTS IN 28 CASES OF MENINGIOMA AT THE CEREBELLOPONTINE ANGLE.

RESULTS	Number	Per cent (of 28)
Normal labyrinths, bilateral	5	18
Slightly hypoactive labyrinth on side of lesion	1	4
Moderately hypoactive labyrinth on side of lesion	1	4
Severely hypoactive or nonfunctioning labyrinth on side of lesion	19	68
Nonfunctioning labyrinths, bilateral	1	4
Moderately hypoactive labyrinths, bilateral	1	4

Caloric tests for evaluation of the function of the equilibrial labyrinths were carried out in 28 cases (see Table 2). Equilibrial labyrinthine function was normal in five (18 per cent) of these 28 cases. In most of the entire series, a pronounced loss of hearing was accompanied by a correspondingly severe decrease in equilibrial labyrinthine function; however, labyrinthine function was normal in two instances of severe deafness on the side of the meningioma, and only a slightly damaged equilibrial labyrinth was found in one instance of extensive ipsilateral decrease in auditory function.

SIGNS AND SYMPTOMS SUGGESTING INTRACRANIAL ORIGIN OF VIIITH-NERVE DYSFUNCTION.

Detailed evaluation of signs and symptoms referable to the VIIIth cranial nerve is essential, but they must be accompanied by evidence of changes in the central nervous system in order to place the lesion at the cerebellopontine angle and to suggest that the tumor is a meningioma.

Table 3.

FINDINGS IN ADDITION TO DYSFUNCTION OF THE VIIITH NERVE IN 55 CASES OF MENINGIOMA AT THE CEREBELLOPONTINE ANGLE.

SIGNS OR SYMPTOMS	Number	Per cent (of 55)
Corneal anesthesia	40	73
Ipsilateral only	27	50
Contralateral only	1	2
Bilateral	12	22
Paresthesia	27	49
Face	19	35
Tongue	6	11
Extremities	6	11
Bilateral	4	7
Headache	32	58
Frontal	7	13
Occipital	13	24
Generalized	12	22
Facial weakness	15	27
Slight	10	18
Moderate	4	7
Complete	1	2
Bilateral	2	4
Spontaneous nystagmus	45	82
To right and left	40	73
Toward side of lesion only	3	5
Toward side opposite lesion only	2	4
Vertical component also	21	38
Facial pain	3	5
Trigeminal neuralgia	3	5
Visual disturbances	19	35
Choked disk	23	42
Disturbances in gait	39	71
Difficulty in swallowing or defects in speech	15	27
Weakness of pharyngeal muscles	6	11
Paralysis of vocal cord	3	5
Positive X-ray findings	15	27
Suggesting cerebellopontine-angle lesions	6	11
Suggesting increased intracranial pressure	9	16

Table 3 lists the symptoms and signs of involvement of the central nervous system which, taken in conjunction with those of decreased function of the VIIIth cranial nerve, indicated pathologic changes at the cerebellopontine angle. Spontaneous nystagmus was the commonest finding that, in addition to evidence of VIIIth-nerve dysfunction, suggested a lesion in the posterior fossa. This abnormality was found in 45 of the 55 patients (82 per cent). Three patients presented spontaneous nystagmus with the quick component toward the side of the lesion only; in two patients, the quick component was present only when the eyes were deviated toward the side opposite the meningioma. In the great majority of instances (40, or 89 per cent of the afore-mentioned 45 patients), the spontaneous nystagmus was present on lateral gaze both to the right and to the left; in 21 instances, a vertical component to the spontaneous excursions of the eyes also was present.

The spontaneous nystagmus associated with cerebellopontine meningiomas was more often coarser and slower toward the side of the lesion than toward the unaffected side. The quick and slow components of the nystagmus were not so clear-cut as in the usual labyrinthine nystagmus. Although in most instances they were not present when the patient was looking straight forward, the ocular excursions became wild and active on deviation of the eyes from the midline. The nystagmus of the two eyes was commonly in perfect rhythm and, although it was especially vigorous, it was easily reversed by caloric stimulation of the ear.

Corneal anesthesia was the second commonest additional finding suggestive of intracranial disease (40 instances, or 73 per cent). The decreased corneal sensitivity most often was on the side of the lesion only, but it was bilateral in 12 of 40 instances. The corneal anesthesia frequently was pronounced and easily demonstrated, but repeated examinations were necessary at times to establish the decreased sensitivity.

Headache was a common finding. Although considerable variation occurred in the location and type of this discomfort, it was often a major complaint and frequently caused the patient to seek medical assistance. The headache ordinarily tended to increase in severity from the time of its onset and differed sufficiently from the usual type of cephalic pain to cause the clinician to seek accompanying findings suggestive of serious organic disease.

Twenty-seven (49 per cent) of the patients presented some type of paresthesia. The face or tongue was involved most frequently, and usually the sensation was constant and unilateral and progressed to involve adjacent regions strongly suggesting the organic nature of the complaint; however, this symptom was less definite in a few instances. One patient complained that numbness had been present in the ring finger of each hand for several months. A second patient had noticed a prickling sensation of the finger tips for one year. Another had experienced numbness of the left hand for 18 months, and the sensation gradually had extended to involve the ipsilateral side of the body and the ipsilateral leg and foot.

Fifteen patients presented some degree of facial weakness. In most instances, paresis was slight and evident only on close inspection. Bilateral involvement of the facial nerve was present in two patients.

Visual disturbances were frequent (19 patients, or 35 per cent). Failing vision, diplopia or blurring of vision were the usual complaints which, along with other findings, led to ophthalmoscopic evaluation and the finding of choked disks in 23 (42 per cent) of the patients.

As noted in Table 1, some type of dizziness was present in 22 instances (40 per cent) and most frequently was postural in nature. True spinning vertigo was found in six instances; in every case in which the vertigo possibly might be confused with labyrinthine imbalance, additional findings were present that pointed toward an intracranial origin of the vertigo. One patient complained of mild postural imbalance of six months' duration and on one occasion experienced violent spinning vertigo on lying down. The vertigo lasted only a few minutes, was not accompanied by nausea or otic symptoms and had been preceded by rapidly increasing occipital headaches. A second patient gave a history of two short attacks of subjective vertigo that were preceded by paresthesia of the face.

A third patient noticed that sudden turning to the right would produce a short attack of vertigo once or twice daily. She also had been conscious of numbness of the ipsilateral angle of the mouth for six years. A fourth patient had been subject for three years to sudden short attacks of vertigo not initiated by positional change. She invariably fell to the right side (opposite the lesion) and had injured herself several times. Burning of the ipsilateral side of the tongue was present before the onset of the attacks of vertigo, and she was frequently unconscious during these episodes. While playing volleyball, a fifth patient fell to the ground because of a violent attack of vertigo that lasted 15 minutes. He gave a history of increasingly severe headaches and on examination presented a central type of spontaneous nystagmus.

Helpful roentgenologic findings were present in 15 of the patients (27 per cent). Destruction of the sella turcica was present in seven instances, erosion of the tip of the petrous pyramid was evident in three, and the tumor had caused decalcification of the tip of the petrous pyramid in two instances. The posterior clinoid processes were eroded in two cases, and a large irregular calcified tumor in the posterior fossa with extensive secondary erosion from pressure of the sella turcica and the tip of the petrous pyramid was present in one case. The other findings in Table 3 do not call for further elaboration.

In order to complete the findings in this series of meningiomas at the cerebellopontine angle, it appears pertinent to present one case in some detail.

A 43 year old woman had noticed the onset of right tinnitus and deafness eight years before reporting for examination The deafness rapidly became severe Nine months before she was examined, chills and fever had been present for one day At this time she had noted a period lasting from five to ten minutes during which there was severe right frontal headache accompanied by visual hallucinations (spinning disks before the right eye only) Eight months before examination, the patient had discovered that she could touch a growth in her right ear with a hairpin There had been no discharge from the ear Examination disclosed a warty tumor almost completely occluding the right external auditory canal and complete right deafness Further evaluation demonstrated bilateral choked disks and an eroded sella turcica Biopsy of the mass in the external auditory canal established a diagnosis of meningioma

COMMENT.

Table 4 presents a comparison of the symptoms and signs in these 55 cases of meningioma at the cerebellopontine angle with the symptoms and signs in 150 cases of unilateral acoustic neuroma that have been studied previously. Differential points shown in the table will be amplified.

Table 4.

RELATIVE FREQUENCY OF SYMPTOMS AND SIGNS IN MENINGIOMAS AT THE CEREBELLOPONTINE ANGLE AND IN ACOUSTIC NEUROMAS.

SYMPTOMS	Cerebellopontine angle meningioma (55 cases)		Acoustic neuroma (150 cases)	
	Number	Per cent	Number	Per cent
Loss of hearing or tinnitus as first symptom	35	64	105	70
Severe or total loss of hearing on side of lesion	33	60	114	76
Moderate loss of hearing on side of lesion	10	18	19	13
Slight loss of hearing on side of lesion	6	11	2	1
Normal hearing	6	11	0	0
Tinnitus	29	53	100	67
Vertigo	6	11	16	11
Dizziness	22	40	53	35
Corneal anesthesia	40	73	126	84
Ipsilateral only	27	50	110	73
Bilateral	12	22	16	11
Paresthesia	27	49	77	51
Headache	32	58	105	70
Facial weakness	15	27	55	37
Slight to moderate	14	25	9	6
Complete	1	2	1	1
Spontaneous nystagmus	45	82	125	83
Choked disk	23	42	74	49
Facial pain	3	5	16	11
Trigeminal neuralgia	3	5	0	0
Visual disturbances	19	35	47	31
Positive x-ray findings	15	27	64	43
Positive Romberg	39	71	106	71

Meningiomas are slow-growing vascular tumors that choose certain special regions of the meninges as their point of origin and exert their harmful influence by the production of gradually increasing pressure on the surrounding intracranial structures as well as the contiguous calvarium or the base of the skull. These tumors almost never metastasize and, as already mentioned, may attain great size before they produce recognizable clinical symptoms.

The cerebellopontine angle is one of the favorite points of origin for meningiomas. Here the crowded structures of the brain stem and its attached cranial nerves, together with the cerebellum, are gradually pushed aside and distorted until their functional parts are sufficiently damaged to result in increased intracranial pressure, signs and symptoms of changes in the cranial nerves, and cerebellar incoordination. As the tumor encroaches on the petrous portion of the temporal bone, decalcification or bony destruction is brought about. If there is extensive involvement of the temporal bone, the tumor may reach the middle ear or the external auditory canal and be evident on direct inspection.

The VIIIth cranial nerve appears to be particularly prone to injury by tumors originating at the cerebellopontine angle, and meningiomas found in this region are no exception. Loss of hearing, tinnitus and disturbed equilibrium are usually present, are commonly prominent and, in the early stages, may be the only symptoms that lead the patient to consult his physician. The neural type of loss of hearing found in meningiomas at the cerebellopontine angle most commonly progresses rapidly to severe or total lack of cochlear function. In this series, 33 patients (60 per cent) presented insufficient remaining hearing at the time of their initial examination to be suitable subjects for audiographic study and special tests of hearing; however, only slight to moderate deafness was found in 16 instances (29 per cent). It is in this latter group of patients, who have considerable residual hearing, that the diagnosis is likely to be obscure and that tests for recruitment are of value in differentiating the end-organ type of nerve deafness from hearing loss due to injury to nerve fibers at the cerebellopontine angle. As noted previously, tests for recruitment were done on two patients with meningiomas at the cerebellopontine angle and recruitment was absent in both.

Six of these 55 patients demonstrated that normal hearing in the presence of a lesion obviously situated at the cerebellopontine angle does not necessarily rule out meningioma. Loss of hearing or tinnitus is the commonest initial symptom produced by meningiomas of the cerebellopontine angle, the incidence being 64 per cent in this series. Commonly, additional findings soon indicate the deafness to be of intracranial origin. Twenty-nine of 46 patients in this series noticed symptoms other than deafness within four years. Infrequently, however, deafness or tinnitus may be the only symptom for many years, as demonstrated by the afore-mentioned patient who had impaired hearing, as well as tinnitus, for 25 years before the appearance of additional symptoms suggesting pathologic changes at the cerebellopontine angle. A meningioma at the cerebellopontine angle is a possibility in any instance of unexplained nerve deafness, particularly if the loss of hearing is unilateral and rapidly progressive. More serious consideration must be given to this lesion if recruitment is absent and there are additional findings suggesting pathologic changes in the posterior fossa.

Varying degrees of equilibrial disturbance are common in meningiomas at the cerebellopontine angle; however, such a lesion is an extremely remote possibility if true spinning vertigo is present and there are no additional symptoms, such as paresthesia, spontaneous nystagmus, visual disturbances or unusual types of headache.

Normal audiograms and normal results of caloric tests do not rule out meningioma at the cerebellopontine angle. Evaluation of decreased cochlear and labyrinthine function, when considered in conjunction with additional signs and symptoms, may suggest the correct diagnosis. A careful history is essential, and particular inquiry should be made regarding equilibrial disturbances, headaches, paresthesia, diplopia, blurred or failing vision, facial pain, difficulty in swallowing, defects in speech and hoarseness.

Roentgenograms of the head may indicate local changes due to pressure on contiguous bony structures, evidence of increased intracranial pressure, or a calcified tumor suggestive of meningioma.

Examination of the ocular fundi is always important in unexplained nerve deafness, as choked disks suggest an intracranial origin of the loss of hearing.

Search for evidence of involvement of cranial nerves is extremely important in suspected meningioma at the cerebellopontine angle. Involvement of the Vth nerve, with corneal anesthesia or decreased sensation over the face, is a frequent finding. Facial weakness on the side of the lesion may be present, although it is seldom pronounced and only occasionally complete.

Paralysis of the palate or the constrictor muscles of the pharynx, deviation of the tongue, and nonfunction of the ipsilateral vocal cord may be present, being noted most commonly in far-advanced meningiomas at the cerebellopontine angle.

Findings indicative of involvement of cranial nerves are usually noted on the same side as the meningioma but, due to torsion of the brain stem or direct pressure from the tumor, these findings occasionally may appear on the side opposite the lesion.

Examination of Table 4 demonstrates that the signs and *symptoms of meningiomas at the cerebellopontine angle are similar to those of acoustic neuromas originating in the same region. In many instances, it is impossible to differentiate preoperatively between these two lesions.*

Since acoustic neuromas at the cerebellopontine angle are so much commoner than are meningiomas, a tumor in this region must be considered to be a neuroma until certain atypical features, such as the following, suggest the presence of a meningioma:

1. Labyrinthine function almost always is impaired or completely abolished in acoustic neuromas, whereas it is normal in a small percentage of meningiomas at the cerebellopontine angle.

2. Meningioma is a more likely possibility than is acoustic neuroma in the presence of normal hearing and a tumor that is obviously situated at the cerebellopontine angle.

3. Meningioma is to be considered as a possibility in any instance of a tumor at the cerebellopontine angle with slight to moderate loss of hearing.

4. With severely depressed hearing and normal or slightly decreased labyrinthine function, meningioma is more probable than is acoustic neuroma.

5. Meningioma is the more probable diagnosis if trigeminal neuralgia is one of the findings associated with a tumor at the cerebellopontine angle.

6. Roentgenographic examination may show enlargement of the internal auditory meatus, which suggests acoustic neuroma, or it may disclose a calcified tumor or changes in the surrounding skull, findings that are characteristic of meningioma.

7. The tumor occasionally may be present in the external auditory canal or middle ear and the differential diagnosis may be made by microscopic examination of tissue.

SUMMARY AND CONCLUSIONS.

A review was made of 55 cases of meningioma at the cerebellopontine angle in which operations were performed and in which the diagnoses were confirmed by microscopic examination of tissue. An attempt was made to list signs and symptoms that, when found in addition to evidence of impaired function of the VIIIth cranial nerve, were suggestive of a lesion at the cerebellopontine angle. An attempt also was made to determine whether there were any signs or symptoms peculiar to meningiomas at the cerebellopontine angle that might tend to allow differentiation of this lesion from the commoner acoustic neurofibroma.

On the basis of this study, the following conclusions are made:

1. Meningiomas, while not common, are of sufficient frequency to be considered in the differential diagnosis of most lesions obviously situated at the cerebellopontine angle.

2. Signs and symptoms indicating disturbed function of the VIIIth cranial nerve usually are prominent and early in meningiomas at the cerebellopontine angle, and detailed evaluation of hearing and of equilibrial labyrinthine function is valuable in reaching the proper diagnosis.

3. Evaluation of the function of the VIIIth cranial nerve may suggest a lesion at the cerebellopontine angle, but the findings must be linked with signs and symptoms of intracranial pathologic changes before the diagnosis of a tumor in this region can be established.

4 Preoperative differentiation of meningioma and acoustic neuroma is impossible in many instances, but at times the otologist can offer substantial assistance in the distinction between these two lesions.

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THE TRIAD OF KARTAGENER. RELATION OF UPPER TO LOWER RESPIRATORY PATHOLOGY.

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I. INTRODUCTION.

The intimate relationship between the disease of the upper and lower respiratory airways has become more and more evident in recent years through radiological and bronchographic studies and a more careful evaluation of the patients with respiratory symptomatology.

It is not unusual for the chest physician in the diagnosis and therapy of pulmonary diseases to be confronted with symptoms such as cough, hemoptysis, expectoration, dyspnea and wheeze whose origin relates to pathologic conditions of the upper respiratory passages. On the other hand, the otolaryngologist treats conditions of the nose and throat that have their origin in the lungs.

Cracovaner[1] has recently analyzed the principal symptoms of pulmonary disease related to nose and throat pathology. Cough, in particular, may be reflex from areas in the nose due to irritation from mucus, congestion or fumes, enlarged turbinates, or from contact due to a deviated septum. Infections, nasal allergy and vasomotor disturbances play an important role in this reflex.

In children, infection of the tonsils and adenoids may predispose to exacerbations of cough. The cough that accompanies

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a recurrent naso-pharyngeal condition or persists after an upper respiratory infection is frequently associated with an allergic state: sensitivity to bacteria in the excessive lymphoid tissue. This condition may account for the asthmatic component of infections and allergic conditions of the nose and paranasal sinuses.

The anatomical consideration that the mucous membrane of the upper respiratory tract is continuous with that of the trachea and bronchi explains how readily an infective process can be transmitted from one segment to another. On a physiopathologic basis the upper respiratory tract influences the lower respiratory system as an air conditioner of the inspired air, originating naso-broncho-pulmonary reflexes, as a source of germs for the lower tract and initiating phenomena of hypersensibility in the respiratory system. Similarly, a pathologic condition of the tracheo-bronchial tree may influence the nose and paranasal cavities.

Clinically, the relationship between sinusitis and pulmonary conditions, particularly bronchitis and bronchiectasis, is of prime importance.

Price[2] studied the frequency of sino-bronchitis among infants and children with symptoms of chronic respiratory tract infection and among those with no respiratory symptomatology. Allergy seems to be an important factor in childhood sino-bronchitis. Roche[3] studied the etiology and pathogenesis of rhino-bronchorrhea and bronchiectasis in infancy. On the other hand, there are descriptions of sinus infection in children that fail to recognize the frequent association of sinusitis and broncho-pulmonary disease.

Mounier-Kuhn[4] described in children the syndrome "ethmoidoantritis and bronchiectasis" as manifest at the highest and lowest level of an affection involving the entire respiratory system. An allergic background seems to be present in most of these patients.

These observations pose the problem of the pathogenesis of bronchiectasis as congenital or acquired, primary or secondary to the upper respiratory condition, which has been discussed from a clinical point of view, but seldom on a pathologic basis.

In 1933, Kartagener[5] correlated etiologically a congenital anomaly to the disorders of the upper and lower air passages and described a syndrome consisting of complete transposition of the viscera, bronchiectasis and chronic rhino-sinusitis. The usual findings in the upper respiratory tract are nasal polyposis, chronic hyperplastic rhinitis, ethmoido-maxillary sinusitis and absence or underdevelopment of the frontal sinuses.

A review of the literature revealed that more than 100 cases of Kartagener's syndrome have been reported; however, not all of the cases show an undisputable radiological evidence of bronchiectasis and a complete *situs inversus.* For this reason, Dickey[6] has recently suggested to substitute dextrocardia for complete transposition of viscera.

The clinical, radiological and pathological study of two cases of Kartagener's syndrome prompted the evaluation of the relationship between the several items of this syndrome, particularly between upper and lower respiratory pathology.

II. CASE REPORTS.

Case 1: M. L. B., 19-year-old white male was seen in the ENT Clinic, Research and Educational Hospitals of the University of Illinois because of a persistent cough, drainage from the nose and absence of the sense of smell. The past history revealed a chronic cough, productive of a thick, yellowish sputum, as long as he could remember; however, it had been only since three years before, at which time the patient fell into a ditch, that the cough became troublesome and more productive, and at present about one cup of this thick, yellow foul smelling sputum is brought up daily. His nose has been discharging as long as the patient can recall; first of a thin colorless, watery type and subsequently of a thick, purulent nature. He had never known a sense of smell, though the odor of his discharge had been described to him as bad. Nasal polyps had been removed in the past, but without any result. Family history was noncontributory.

Physical examination revealed a well-developed white male, apparently not acutely ill. There was a fusiform swelling of the joints of both hands and feet interpreted as rheumatoid arthritis. Examination of the chest and abdomen revealed a complete transposition of the viscera. Hypoventilation was noted in the right plumonary field, while moist rales were heard in the left lower segment of the lung.

The otolaryngological examination revealed a considerable amount of muco-purulent secretion in both middle meatuses, nasal floor, nasopharynx and posterior wall of the oropharynx. Both inferior turbinates were quite large and polypoid in appearance. The tympanic membranes were opaque, dull and lusterless, but without any evidence of perforation or scar. The larynx was essentially normal. The transillumination revealed darkness of all the sinuses. An irrigation of both antra through the natural ostia revealed a considerable amount of foul, purulent discharge.

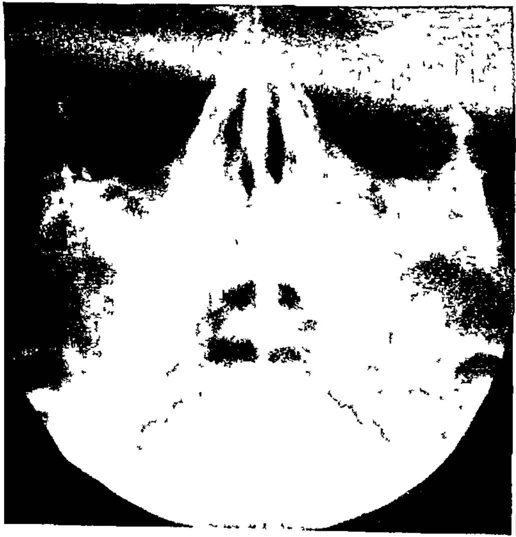

Fig. 1 Routine Waters' projection revealed diffuse clouding of both antral sinuses. This was a part of a pansinusitis.

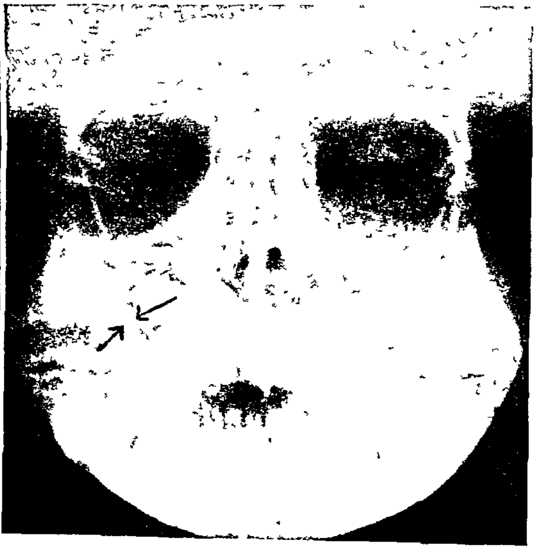

Fig. 2 Right antral sinogram reveals *irregularity* and deformity of the sinus cavity by thickened hyperplastic mucosa. Note the *space between the lateral margin of the* opaque material and the lateral bony wall of the antrum.

The X-rays of the sinuses showed the frontal sinuses to be poorly developed and cloudy; their walls lacked the usual sharp white cortical line (see Fig. 1). There was diffuse clouding of both antral and sphenoid sinuses and partial opacification of the ethmoid cells, indicating pansinusitis.

A right antral sinogram showed the deformity of the contrast material due to hyperplastic antral mucosa (see Fig. 2). From the appearance of the plain films there is reason to believe that a similar condition is present on the left side.

The X-rays of the temporal bones revealed normal development and pneumatization of the mastoid processes.

In the chest X-ray the cardiovascular shadow was noted to be reversed—the apical region appearing prominent on the right side instead of the left, as in the usual orientation. This is a part of a complete *situs inversus*. The plain chest film further revealed increased radiolucency in the right lower lung field compatible with emphysema. An oblique density is seen through the heart shadow and may represent an atelectatic lower lobe. A diffuse reticulated increased density, suggesting honey-combing, was noted in the left lower lung field. These changes are probably all secondary to bronchiectasis (see Fig. 3).

The upper and lower gastro-intestinal study revealed all the viscera to be completely reversed, establishing the presence of a complete *situs inversus*.

The X-rays of the limb joints revealed normal feet, shoulders and elbows. Soft tissue swelling about the middle interphalangeal joints with equivocal cystic changes in the periarticulatory surfaces were interpreted as early rheumatoid arthritic changes.

A thorough laboratory work-up was done. The standard electro-cardiogram revealed typical "mirror image" tracing of dextrocardia. Urinalysis was normal. Serology was negative. Complete blood count showed a white count of 11.450, otherwise normal. Blood chemistry was within normal limits. Sedimentation rate (Wintrobe corrected) was 28 mm. per hour. Sputa were negative for acid fast bacilli, but positive for Neisseria catarrhalis, alpha streptococci and Friedlander's bacillus.

The allergy consultation reported history of nasal polypectomy, but no history of sensitivity to food, drug, vegetation, dust or animals, and no previous apparent allergic trouble. Intracutaneous tests to all the available exogenous allergens were negative. Treatment with antihistamins gave no result.

The bronchoscopy showed the mucosa of the tracheo-bronchial tree to be moderately hyperemic and edematous. A profuse amount of milky secretions gushed forth from all the orifices. It appeared that the main bronchi were reversed in position.

The bacteriological report of bronchial secretions revealed Neisseria catarrhalis, alpha streptococci, hemophilus influenzae and diphtheroids.

The bronchogram revealed the lungs to be transported, since an upper, middle and lower lobe bronchial branch was demonstrated on the patient's left side and an upper including lingula and lower lobe branch was outlined on the patient's right side.

Tubular dilatations of the lower lobe bronchial tree on the left supported the plain film impression of left lower lobe bronchiectasis (see Fig. 4). Other films, not shown, demonstrated that the right lower lobe bronchial tree could not be visualized beyond approximately a 3 cm. stump, again supporting the probability of right lower lobe collapse and compensatory emphysema of the upper lobe.

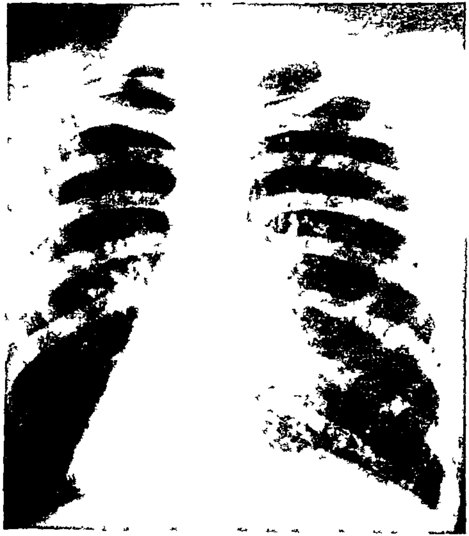

Fig. 3. Chest. Note the reversed *orientation* of the cardiovascular structures. This was part of a complete situs *inversus, also* note the irregular increased markings above the left diaphragm and extending into the left hilar and perihilar area. The pulmonary changes are bronchiectatic in nature.

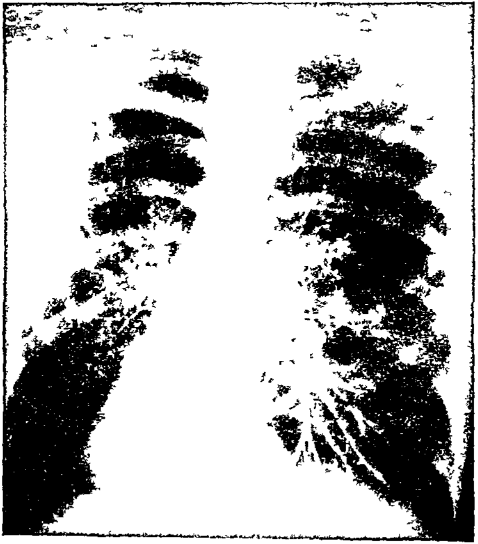

Fig. 4. Bronchogram. Note the tubular dilatation of the lowermost bronchi representing *bronchiectasis*.

A right lower lobectomy was, therefore, decided upon. At surgery the right upper lobe was enormous, while the lower was a small shrunken atelectatic dark colored structure, which lay far posteriorly.

The pathologic diagnosis was bronchiectasis of the right (left) lower lobe with bronchostenosis. Microscopically, the sections through the lung showed dilated bronchi, lined by ciliated columnar epithelium. The bronchi were surrounded by an increased amount of fibrous tissue and areas of focal and diffuse lymphocytic infiltration. The cystic lumina were filled with acidophilic material and mononuclear cells. Some of these cells were large; there was also multinucleated giant cells. Adjacent lung parenchyma showed the alveoli to be filled with red blood cells, mono- and polymorphonuclear cells. There were areas where the alveoli were collapsed. The trabeculae appeared to be thickened and showed an increased amount of fibrous tissue.

A naso-antral window was performed bilaterally at a later date to improve sinus drainage. Marked relief of cough and nasal discharge followed these two surgical procedures; however, sputum, though reduced considerably, did not clear up completely. Repeated Proetz displacements turned out to be quite useful to facilitate his sinus drainage.

Case 2: D. D., 39-year-old colored female, came to the ENT Clinic of the Research and Educational Hospitals of the University of Illinois because she had had "a cold in her head and chest all her life." She used two large boxes of tissues per week to blow her nose, which produced white to yellow discharge. She could not smell because of her trouble. Her past history revealed that she had productive cough and excessive nasal discharge ever since she could remember. She had been coughing day and night; sputum was thick, yellow or greenish and approximately one cupful a day. No hemoptysis nor chest pain. Nasal discharge was excessive and there was stuffiness in her nose. No epistaxis nor headache. She had lost 13 pounds in ten months.

Tonsillectomy and adenoidectomy was done in childhood. She had some treatment for her sinuses, and antrostomies were performed a few years earlier on both sides.

Family history revealed that her father died 18 years before of tuberculosis. Her brother has been treated all his life for bronchiectasis and sinusitis, but has no transposition of viscera.

Physical examination revealed a well-developed, moderately nourished colored female with a mild finger clubbing. Complete transposition of the viscera was noted on examining the chest and abdomen. Moist rales were heard in both lower pulmonary fields.

The otolaryngological examination revealed muco-pus in both middle meatuses, nasal floors and naso-pharynx. The middle and lower turbinates appeared very large and lined by a boggy and pale mucosa. The tympanic membranes were dull and lusterless, with no sign of perforation or healed perforation. The larynx was normal. The transillumination revealed opacity of all the sinuses.

The X-rays of the sinuses revealed the frontals to be undeveloped. There was clouding of the left antrum, and membrane thickening of the right antrum (see Fig. 5). The ethmoid and sphenoid sinuses appeared involved, too. These findings indicate pansinusitis. A left antral sinusogram confirmed the evidence of membrane thickening and antral sinusitis (see Fig. 6). The X-rays of the mastoids showed normal pneumatization of these processes.

The plain films of the chest revealed *situs inversus* and diffuse mottled increased densities in the left lower lung field. There was a suggestion of "honey-combing," indicating that this is a diffuse inflammatory

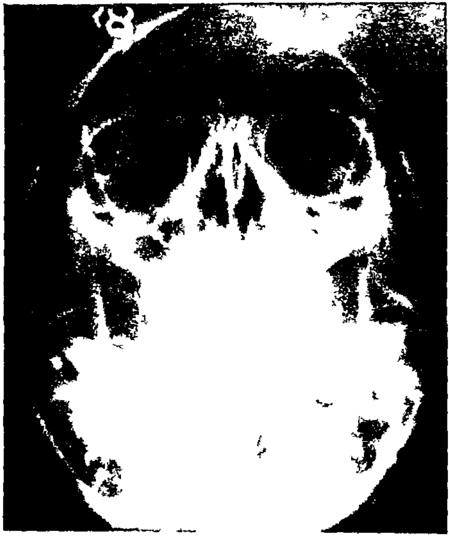

Fig. 5. Sinus examination Waters View. Note the diffuse clouding of the left antral sinus plus the membrane thickening in the right antral sinus *secondary to sinusitis*.

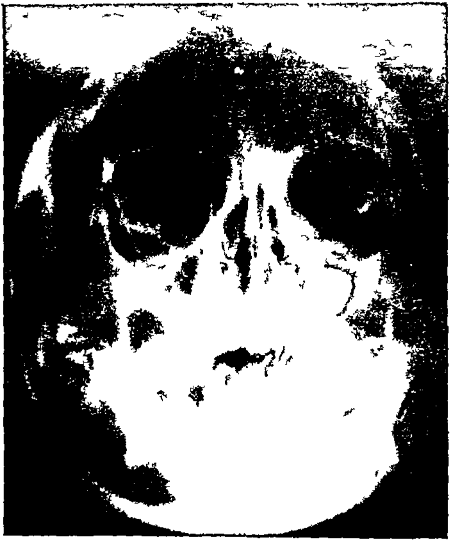

Fig. 6. Sinogram of *left antrum*. Note the globular appearance of the contrast material within the sinus associated with mucosal thickening.

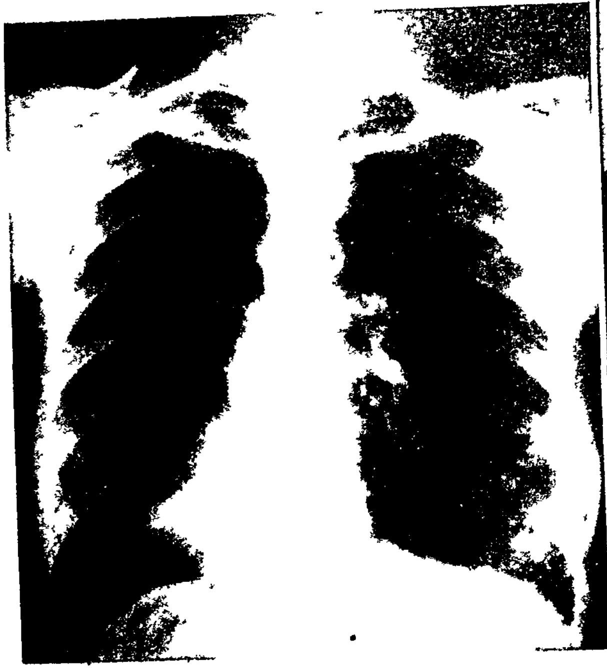

Fig. 7. Chest. Note the dextrocardia and right-sided position of stomach, a part of the patient's complete situs inversus. Also note the irregular mottled appearance of the lower lung field, the left in particular. These were due to bronchiectasis.

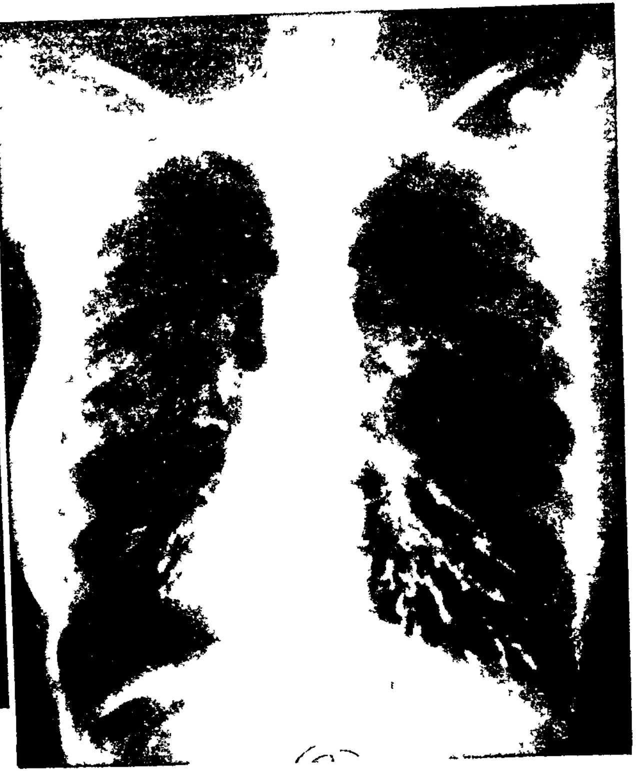

Fig. 8. Bronchogram. Note the bilateral cylindrical and saccular changes in the lower bronchial tree secondary to bronchiectasis.

process with possible underlying or superimposed bronchiectasis These changes extended well into the base of the left lung The right lower lung field contained numerous areas of increased radiolucency between the normal pulmonary markings The basilar area looked emphysematous probably bulbous or bleb formation An oblique line seen through the heart shadow and extending roughly to the spine suggested partial collapse of the right lower lobe (see Fig 7)

The upper and lower gastro intestinal study confirmed the presence of a complete *situs inversus*, otherwise it appeared normal

The reports of the laboratory work up confirmed the diagnosis The standard electrocardiogram showed the typical tracing of dextrocardia Urinalysis was normal Serology was negative Complete blood count was normal except for a slight increase in the white cells Blood chemistry was within normal limits Sedimentation rate (Wintrobe corrected) was 38 mm per hour Sputum was negative for acid fast bacilli but showed Neisseria alpha streptococci hemophilus influenzae

An allergy survey with intracutaneous tests failed to reveal any allergic etiology of her respiratory symptomatology

A bronchoscopy revealed both main bronchi to be filled with copious thick tenacious muco pus Hyperemia involved all tracheobronchial tree The disposition of the bronchi confirmed the *situs inversus* Impression was that of severe bilateral bronchiectasis involving chiefly the basilar segments The bronchial culture revealed many hemophilus influenzae a few Neisseria and alpha streptococci

A bronchogram confirmed the presence of bilateral tubular and cystic bronchiectasis involving the entire lower lobe on both sides with probable collapse of the dorsal basal segment of the patient s right The lingula on the right side was also involved (see Fig 8)

Due to the extensive involvement of the lungs surgical treatment was not deemed advisable Proetz irrigations resulted in relief of her sinus drainage Acute flare ups of the condition were adequately controlled with antibiotics and inhalations

III PATHOGENESIS OF KARTAGENER'S TRIAD

Various views on the nature of the relationship between the items of Kartagener's syndrome have been advanced by several authors

The transposition of viscera is a rare congenital anomaly characterized by a disturbance in the bilateral symmetry of the body Its occurrence is about 1 to 10,000 (Kartagener,[7] Adams and Churchill, Torgersen,[8] Pavlitzek[9]) among the general population

According to the law of multiple anomalies, several other congenital anomalies have been reported with *situs inversus* In particular, cases of Kartagener's triad have been described with an anomalous left subclavian artery,[10] malformations of the retinal vessels,[11] renal anomalies,[1] cardiovascular defect,[6, 13]

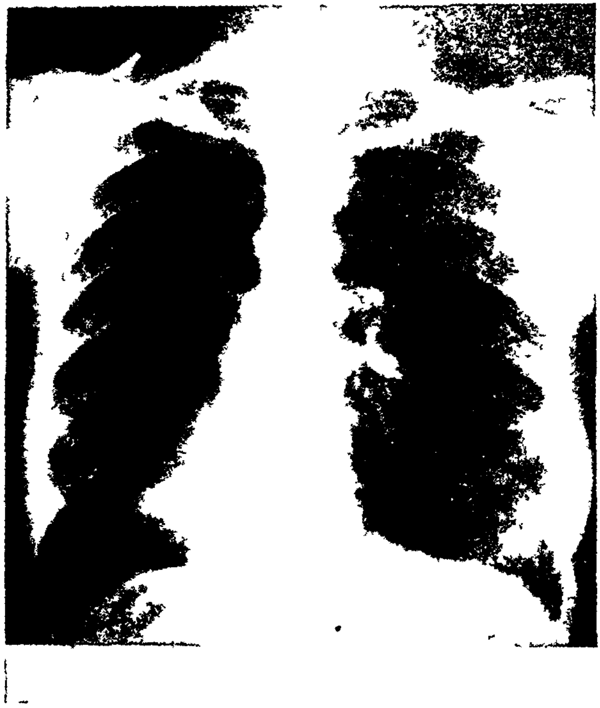

Fig. 7. Chest. Note the dextrocardia and right-sided position of stomach, a part of the patient's complete situs inversus. Also note the irregular mottled appearance of the lower lung field, the left in particular. These were due to bronchiectasis.

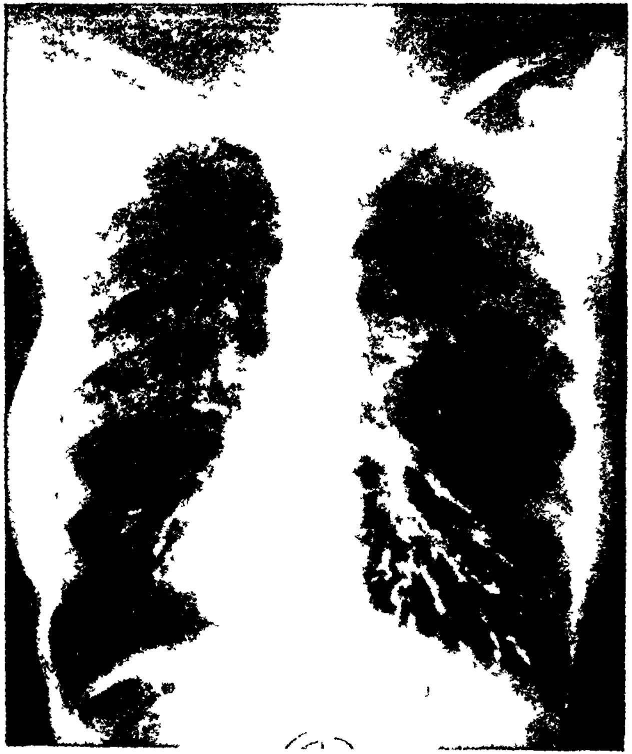

Fig. 8. Bronchogram. Note the bilateral cylindrical and saccular changes in the lower bronchial tree secondary to bronchiectasis.

In an analysis of the pathogenesis of bronchiectasis, Sebesteny[27] considers three factors, the development of the lung, functional disturbances of the bronchial system and infection. Recent studies have shown that the development of the lung by differentiation, budding and expansions of pulmonary anlages continues after birth for approximately seven years. Defective postnatal lung development has been observed in early life associated with metabolic, endocrine, reticulo-endothelial and connective tissue disturbances. Bronchiectasis may develop in those pulmonary segments that remain in their fetal collapsed state and do not expand after birth. In fact, alveoli do not form in the atelectatic segments, and the corresponding bronchi, unsupported externally by aerated alveoli, may dilate as a result of the atmospheric pressure. The mucus secreted may become infected and contribute to the bronchial weakening and dilatation.

It is conceivable on the other hand that atelectasis may develop in a lobe previously normal with resultant bronchiectasis. The study of a large series of X-ray films has shown that collapse of a lobe or a segment of a lobe is quite frequent in lower respiratory infections, and that there is a tendency for dilatations of the bronchi and bronchioles supplying the atelectatic area. Failure of these bronchi to return to normal when atelectasis supersedes accounts for the development of bronchiectasis. It is at this moment that the local factors act to determine the persistence of dilatation of the bronchi. In both of our cases, lobar or segmental collapse was associated with bronchiectasis of a non-congenital type.

It seems, therefore, that a developmental mishap in the bronchi may account for some of the so-called "congenital bronchiectasis", but it is more probable that a congenital factor, evident in Kartagener's triad, predisposes the neonate with *situs inversus* either to defective postnatal lung growth or to atelectasis, and consequently the development of bronchiectasis. The same constitutional factor could represent the predisposition for the development and persistence of chronic sinusitis.

Radiographically the appearance of bronchiectasis in Kartagener's syndrome is generally tubular or varicose rather

turricephaly,[14] absence of the xiphoid process.[15] Multiple cas
of Kartagener's triad in the same family and among twi
have been reported by several authors.[6,8,10,16,17,18] According
Cockayne[19] the transposition of viscera is dependent upon
single autosomal recessive gene, while Torgersen[8] maintai
that it may occasionally be inherited as a dominant characte
occasionally as a recessive one and sometimes not at all herec
tary.

Relationship Between Situs Inversus and Bronchiectasis.

This problem has been the object of discussion and ma
observations. Some authors favor the congenital theory
origin of bronchiectasis as contrasted to the theory that bro
chiectasis is acquired.

Siewert[20] in 1904 was probably the first to speculate on t
congenital etiology of bronchiectasis associated with *situs i*
versus. The description of Kartagener's syndrome and follo
ing reports of this interesting association have further su
ported this theory.

According to Conway[21] the frequency of bronchiectas
among cases with *situs inversus totalis* is so much greater th
in the general population as to suggest that the bronchiectas
may not be fortuitous, but due to a developmental error. T
values vary between 16.5 and 25 per cent in complete *sit*
inversus in contrast with 0.25 to 0.5 among all patients, a
cording to Kartagener,[5] Adams and Churchill,[7] Olsen[22] an
Gross.[23]

Richards[24] reported a case of Kartagener's triad with r
spiratory symptoms since the age of three days. In the ca
of Blixenkrone-Moeller[25] respiratory symptoms had been pre
ent since birth.

The histological study of specimens from lobectomy reveal
in Ingraham's[26] case "persistence of peribronchial connecti
tissue and underdevelopment of alveolar structures," and
the case of Blixenkrone-Moeller[25] "very high cylindric epith
lium in the abnormally wide bronchi and large accumulatio
of lymphoid tissue in the bronchial walls which suggest a co
genital condition".

In an analysis of the pathogenesis of bronchiectasis, Sebesteny[27] considers three factors, the development of the lung, functional disturbances of the bronchial system and infection. Recent studies have shown that the development of the lung by differentiation, budding and expansions of pulmonary anlages continues after birth for approximately seven years. Defective postnatal lung development has been observed in early life associated with metabolic, endocrine, reticulo-endothelial and connective tissue disturbances. Bronchiectasis may develop in those pulmonary segments that remain in their fetal collapsed state and do not expand after birth. In fact, alveoli do not form in the atelectatic segments, and the corresponding bronchi, unsupported externally by aerated alveoli, may dilate as a result of the atmospheric pressure. The mucus secreted may become infected and contribute to the bronchial weakening and dilatation.

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Radiographically the appearance of bronchiectasis in Kartagener's syndrome is generally tubular or varicose rather than cystic, which are usually congenital. In one of our cases the bronchiectasis appeared typically cylindrical while in the other both fusiform and saccular dilatations were seen.

Most authors agree that pulmonary histological findings fail to show a definite evidence of congenital origin for bronchiectasis.[6,17,21,28] The histopathological study of the specimen from lobectomy in one of our patients did not reveal any structural alteration that could definitely be interpreted as congenital. Taiana et al.[29] recently reported that no difference could be observed in their specimen, from common bronchiectasis of the acquired type.

Relationship Between Transposition of Viscera and Upper Respiratory Tract Abnormalities.

The triad, as described by Kartagener, includes nasal polyposis and paranasal sinusitis, but Olsen[22] described four cases of dextrocardia and bronchiectasis without sinusitis and four further cases of dextrocardia and nasal sinusitis without bronchiectasis. According to Gross[23] sinusitis is not an essential item of Kartagener's syndrome, because of its sequential relationship with bronchiectasis.

The presence of nasal polyps or chronic hyperplastic rhinitis in cases of Kartagener's syndrome has been frequently reported. Torgersen[8] described five cases of *situs inversus* associated with nasal polyps without bronchiectasis. Among the cases presented in this paper, one had the history of nasal polypectomy and showed polypoid degeneration of the inferior turbinates, while in the other there was a picture of chronic hyperplastic rhinitis. The hyperplasia of the antral mucosa is demonstrated in the sinusograms. An allergic basis for this condition has been suggested; however, an allergy survey with cutaneous tests to all the available allergens failed to reveal any sensitivity. Sensitization to the allergens in the bacteria may be responsible for the respiratory infections. Blixenkrone-Moeller[25] tried to support this view, demonstrating allergic lung infiltrations in his case of Kartagener's triad.

It may be significant to note that our patient with the history of nasal polypectomy suffered from rheumatoid arthritis d the same ondition presented in the patient reported by uckerman an *Wurtzerbach.*

Banham,[41] i his case of *Kartagener's syndrome, reported* omplete abse of frontal and *sphenoid sinuses and mastoid* air cells and tra. Maxillary *sinuses, as well as the few* ethmoidal cel ere small. Both tympanic *membranes were* hanging in lo olds. The picture *was interpreted as an ar*rest of devel nt and pneumatization *of the temporal bone* at *birth. Bot cases showed poor development of the fron*tal sinuses, ral pansinusitis *and normal pneumatization* of the masto ocesses.

matory process may involve simultaneously both divisions of the respiratory tract.

Brock[32] maintains that sinusitis may be the primary or predominant lesion between sinusitis and a superficial, transitory condition such as bronchitis; however, when morbid anatomical changes develop as in bronchiectasis one must look for a local determining cause or factor either acquired or congenital. A high incidence of sinusitis in bronchiectasis has been reported clinically, while it seems quite uncommon to see bronchiectasis arise from chronic sinusitis. It has been demonstrated radiologically with contrast media that bronchiectasis is significantly related to sinus infection, probably due to intermittent or continuous production of purulent sputum, partially expelled through the upper respiratory airways. The expired air currents, as demonstrated by Proetz, pass beneath the middle turbinates coming in close contact with the sinus ostia.

The knowledge that sinusitis can cause acute, recurrent or chronic lower respiratory infection will explain how sinus infection may maintain and aggravate infections in the bronchiectatic lung. In this way upper and lower processes work together in the persistence of a diffuse respiratory infection. These considerations are of utmost importance in the management of these cases.

An endogenous basis has also been considered for the sinus-pulmonary relationship, and in particular for the development of bronchiectasis in children and in cases of Kartagener's syndrome.

Torgersen[8] in a series of studies on the genetics of Kartagener's triad indicated that "the lung-nose syndrome is inherited as a dominant trait, *situs inversus* behaving as a manifestation of a pleiotropic effect of this gene". In other words, there is a constitutional interrelation between nasal polyps and bronchiectasis, which is hereditary with a varying probability and mode of manifestation connected with the tendency to *situs inversus*.

It is possible that allergy which is inherited as a dominant trait represents the constitutional basis for the development of bronchial and sinus conditions. The mechanical factor appears to be the most important in producing bronchial dilatations, and Davison[33] maintains that allergy, through the production of viscid secretion and narrowing of the bronchial lumen due to edema, may determine all the factors necessary for the development of bronchiectasis in children; and allergy, according to general clinical experience, is considered an underlying factor in the onset of nasal polyps and hyperplastic sinusitis.

Van de Calseyde[34] stresses the synchronism between the hyperplastic sinus pathology and bronchopulmonary disorders. Hypertrophic sinus affection means weakness or disharmony of the normal function and defense mechanisms of the whole respiratory system. Both sinus pathology and bronchiectasis would depend on the same constitutional cause, their reaction to disease varying according to their anatomical characteristics.

In this instance, sinusography appears just as useful in the differential diagnosis of the type of sinusopathy as bronchography in establishing the presence of bronchiectasis; and such differentiation is of utmost importance therapeutically, as fragility of the respiratory system stands for cautiousness in the surgical approach and simultaneous treatment of both pathologic locations.

The management of the sino-bronchial condition in Kartagener's syndrome is, therefore, a corollary to the preceding considerations. A very notable feature of the relationship between upper and lower respiratory pathology is failure of resolution when there is interference with drainage. The greatest effort should be made to control the condition in both segments because of the mutual contribution toward their maintenance. Chronic sinusitis associated with chronic bronchitis is often completely incurable as long as the bronchiectasis is unrelieved. When the bronchiectasis cannot be adequately treated, bronchial drainage should be aided by posture and other physical treatment. When the bronchiectasis is localized, radical surgical treatment is indicated.

Radical sinus surgery is generally contraindicated in association with bronchiectasis. Naso-antral windows, irrigations and Proetz displacement technique are quite useful in alleviating the sinus infection. The control of allergic factors and other systemic disorders is in order. Prompt attention must be given to upper respiratory infections to avoid acute exacerbations of the entire respiratory pathology.

The physician, the rhinologist and the chest surgeon should work as a team in discussing the indications for surgical treatment in these patients and the conservative care in the follow-up.

CONCLUSIONS.

In Kartagener's syndrome, bronchiectasis may represent fetal abnormalities, but more often develop and manifest themselves in the postnatal development of the lung with the concurrence of congenital and environmental factors probably related to an allergic constitution.

Sinusitis seems to develop secondarily, but allergic factors may represent an underlying basis in its inception. This is particularly evident in the presence of nasal polyps and hyperplastic nose and sinus pathology.

Ill-development or underdevelopment of the frontal sinuses seems to be related more to the respiratory pathology than to the transposition of viscera.

A constitutional interrelation between upper and lower respiratory pathology dominates the clinical picture of Kartagener's triad and is fundamental in its therapeutic approach.

SUMMARY.

The intimate relationship between upper and lower respiratory tracts is stressed on anatomical, physiological, pathological, clinical and therapeutic bases.

Two cases of Kartagener's syndrome are reported with the sinographic and bronchographic studies.

The pathogenesis of this syndrome, analyzing the relationship between the items of this triad, is discussed.

The clinical and therapeutic aspects of bronchial disease

and pathology of the upper respiratory tract in Kartagener's syndrome are typical expressions of the unity of the respiratory system. Close cooperation between otolaryngologist and chest physician is a must for therapeutic success

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SIEGLE'S PNEUMATIC EAR SPECULUM MODIFIED FOR A MANOMETER.*

A Preliminary Report on Findings.

RAY M. MOOSE, M.D.,

San Bernardino, Calif.

HISTORICAL.

In his textbook on Diseases of the Ear, written in 1902, Politzer[1] emphasizes "that important method of examination with Siegle's speculum, which is indispensable for the diagnosis of middle ear affections . . ." Through the years the speculum has remained indispensable, and its usefulness has increased, yet there never has been made a fundamental alteration for clinical purposes in that speculum.

WHY MODIFY SPECULUM?

Quite recently, in testing the mobility of the eardrums of hard-of-hearing children using the Siegle speculum, I recorded observations using the terms: *1.* Freely mobile; *2.* Slightly mobile; *3.* Moderately mobile; *4.* Immobile. The question then dwelt with the possibility of a correlation between the amount of hearing and the drum that is slightly mobile, or moderately mobile.

It occurred to me that such terms as *slight* and *moderate* should be eliminated, and that the air pressures in the speculum that make the eardrum move, should be measured. The need, it seemed, was to get measurements of eardrum mobility in various normal and abnormal states of the middle ear and Eustachian tube; therefore, the following modification of the Siegle speculum was made (see Fig. 1).

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* Presented in part when opening the discussion of the paper of Joseph L. Goldman, M.D., "Modern Concepts in Otolaryngological Diagnosis and Therapy," in the Section on Laryngology, Otology and Rhinology of the A.M.A. Annual Meeting, San Francisco, June 23, 1954.

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METHOD AND PROCEDURE.

A hole was drilled into the pneumatic speculum, and into this opening a metal tube about 1 cm. long was welded. Air forced into the speculum can then come out the metal tube and, by means of a rubber tube, can be conducted into a U-shaped mercury or water manometer, or some other type of gauge. Apparently air pressure in the speculum would equally affect the eardrum and the manometer.

The U-shaped manometer is made by bending a glass tube to a rounded "U" and mounting it on a small plastic board, to which is glued a millimeter ruler. Two tubes are so mounted, one to hold mercury, the other water.

With the otoscope thus equipped for measurements and applied to the patient's ear, positive and negative pressures are gradually increased until the umbo (or sometimes a membranous part of the drum) is seen to make its smallest visible movements. While maintaining these movements with pressures as uniform as possible, we see that the column of water is moved up and down the U-tube a total distance of 20 mm. or 25 mm. or sometimes 30 mm. It is then recorded that the umbo moved with static pressure changes of ± 20 mm. to ± 30 mm. water. (Records of total ranges seem more expressive than averages.) To verify observations try pressures up to ± 15 mm. water to make certain the lower pressures do not move the umbo.

PRELIMINARY REPORT.

The principal observations to date are as follows:

1. A normal functioning ear may have a drum that is definitely seen to move with pressures of only 1 or 2 mm. of water. Quite often a posterior segment of the tympanic membrane is seen to move while the umbo remains stationary. It then requires a little more (often 2 or 3 mm. water) pressure to make the umbo move.

2. A person may have normal hearing (not over a 5 db loss for cycles 256 through 8,000) with an ear in which the mobility of the drum is so reduced that the umbo will not move until static pressures of ± 50 to ± 60 mm. mercury are put into the external ear canal. Thus, in answer to the question raised above, there could be no correlation between the threshold of hearing and the mobility of the drum, since normal hearing may also be present with the hard-to-move drum. This is contrary to what is generally expected, as learned from conversations held with different otologists. With one exception, the six or more otologists interviewed said they would expect the ear with the hard-to-move malleus to have impaired hearing.

3. Contrary to what may be expected, the malleus or other parts of the typanic membrane in the ears of some aged individuals are found to move as readily as in the young.

4. Slight congestion of the malleus or other parts of the drum does not seem to hamper its mobility.

5. There seems to be no impairment of mobility after cerumen has been removed. That means ear plugs would not be expected to impair mobility.

6. With acute tubal obstruction, it may take pressures up to ± 60 mm. Hg. to make a small movement of the malleus. After inflation it is gratifying to see the mobility has increased in some cases to 1 mm. or 5 mm. Hg. or less. This correlates with the improved threshold in hearing.

7. Barometric changes change mobility, comparable to acute tubal obstruction. I took a subject with normal ears up a mountain side, and for each 500 or 1000 foot change of elevation, I made observations on the appearance and mobility of the drums. While driving up the mountains the drum was seen to bulge out like an abscess, but retracted when coming down to lower altitudes. This retracted drum was found harder to move than the bulging drum, the umbo of the bulging drum moving with ± 15 mm. to ± 20 mm. Hg., that of the retracted drum requiring ± 25 to ± 35 mm. Hg. pressure. After swallowing, the Eustachian tubes opened, the subject heard better and each eardrum moved with less than 5 mm. Hg.

8. Perhaps of greater interest and practical import are the observations on the following case:

A young man recovering from a cold had severe pain in his right ear during the night. On examination the next afternoon, his right eardrum was red in its entirety. Since the umbo moved with static pressures of only ± 7 to ± 10 mm. water and the posterior quadrant of the drum required pressures of only ± 3 to ± 5 mm. water for visible movements, I decided against a myringotomy at that time. The patient was put to bed in the recovery room, and two hours later the mobility of the drum was again measured. This time the posterior quadrant of the drum was found to be immobile, and the static pressure required to move the umbo had increased threefold. The drum, as yet, was not bulging; however, because of this marked change in mobility it was thought that the middle ear had become partially filled with inflammatory material. A myringotomy, followed by inflation, proved this to be the case.

DISCUSSION.

Sources of Error in Measurements.

It is impossible to keep your eye on the patient's ear and the manometer at the same time. The surge of the mercury, or the water, up and down the U-tube is too rapid for a second party to be of much help. That means that the hand on the air pressure bulb of the otoscope must make uniform pressures, plus and minus, while the eye keeps changing from the eardrum to the manometer. Strictly uniform pressures are impossible, but the recording of the ranges that cause the first visible movements of the drum seems reliable; however, a big easy-to-see excursion of the drum takes a lot more pressure to produce than a movement that is barely visible, and one must be careful to take the reading at the first definite visible movement.

Why Should an Ear With Evident Reduced Mobility of Its Drum Function Normally?

The answer must lie in the fact that the pressures exerted by the pneumatic otoscope do not simulate those of sound waves. They differ as to (a) pressure, as to (b) amplitude, and (c) frequency.

Pressure variations of ± 60 mm. Hg. are equivalent to ± 56,600 dynes per sq. cm., whereas + 980 to – 980 dynes per sq. cm. is the functional range of sound.[2a]

According to Wever and Lawrence[2b] "The amplitude of aerial movements that are of practical importance cover a very wide range, from $10,^{-9\text{ cm.}}$ which is barely perceptible under ideal conditions, to $10,^{-2\text{ cm.}}$ which is approaching a magnitude dangerous to the ear." Now *this amplitude* of vibration, the $10,^{9\text{ cm}}$ is a distance, according to modern physics, that is about one-tenth the diameter of a hydrogen molecule, hence cannot be compared to the eardrum movements made by hand. It is also self-evident that the otoscope cannot move the eardrum with the frequency comparable to audible sound waves having a range of 20 to 20,000 double vibrations per second.

Finally, it is evident that the drum which offers resistance to artificial air pressures in the pneumatic speculum may not necessarily offer resistance to sound waves.

Previous Work on Measuring Eardrum Mobility.

Kobrak[3] did ingenious work on fresh temporal bones measuring linear displacement of umbo from static pressure changes in the outer ear canal in testing eardrum elasticity. Wever and Lawrence[2c] wrote that Dahman, as well as Kobrak, took readings with positive and negative pressures in the ear canal and "obtained greater outward than inward excursions of the drum membrane and manubrium over the main course of measurements." We were unable to find whether the measurements I have described have ever been made as a part of the routine otologic examination.

Is It of Value to Measure Eardrum Mobility in Ear, Nose, and Throat Practice?

The answer is "Yes," but to properly evaluate the various types of otitis media and associated tubal obstructions we need more data. We hope others who are interested will make contributions in this wide open field.

CONCLUSION.

It is a simple procedure to adapt the Siegle ear speculum to a manometer. Eardrum mobility can then be measured, recorded, and communicated as a function of static pressure changes in the outer ear canal. Such measurements take less than one minute of extra time, they give added information, they stimulate interest in the case, and by drawing attention to data observed they call for more work to understand the observations.

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SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

The meeting dates of the Sixth International Congress of Otolaryngology are again emphasized as May 5th through May 10th, 1957. The scientific program for the Plenary Sessions is now complete and is as follows:

CHRONIC SUPPURATION OF THE TEMPORAL BONE.

OPENERS: Marcus Diamant, Central County Hospital, Halmstad, Sweden—Anatomical Etiological Factors in Chronic Middle Ear Discharge.

Luzius Rüedi, Zurich, Switzerland—Pathogenesis and Treatment of Cholesteatoma in Chronic Suppuration of the Temporal Bone.

Horst Wullstein, Director, Otolaryngological Clinic, University of Würzburg, Germany—Surgical Repair for Improvement of Hearing in Chronic Otitis Media.

DISCUSSERS: A. Tumarkin, Liverpool, England; Juan Manuel Tato, Buenos Aires, Argentina; T. E. Cawthorne, London, England; Fritz Zöllner, Freiburg, Germany.

COLLAGEN DISORDERS OF THE RESPIRATORY TRACT.

OPENERS: Hans Selye, Director, Institute of Experimental Medicine and Surgery, University of Montreal, Faculty of Medicine, Montreal, Canada.

Introduction:

Michele Arslan, Padua, Italy—The Upper Respiratory Tract.

Leslie Gay, Physician-in-Charge, Allergy Clinic, The Johns Hopkins Hospital, Baltimore, U. S. A.—The Lower Respiratory Tract.

DISCUSSERS: Victor E. Negus, London, England; Branimir Gusic, Zagreb, Yugoslavia; Aubrey G. Rawlins, San Francisco, U. S. A.; Henry L. Williams, Rochester, Minn., U. S. A.

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OPENERS: Jo Ono, Tokyo, Japan—Etiology.

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Diagnosis:

F. C. W. Capps, London, England—Therapy.

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SUDDEN DEAFNESS OF OBSCURE ORIGIN.*

O. ERIK HALLBERG, M.D.,
Rochester, Minn.

Sudden unilateral or bilateral impairment of hearing is a symptom, not a disease. The deafness under consideration in this report is sudden deafness of obscure cause—not deafness due to trauma, suppurative labyrinthitis, meningitis or mumps. The deafness is alarming to the patient and is almost always associated with roaring tinnitus. True vertigo may or may not be associated with the sudden deafness. If it does occur, it may precede the deafness, come on simultaneously, or come later. Such deafness is seldom or never associated with fatal events or diseases; therefore, factual information about the pathologic process underlying the loss of function is still obscure.

The physician who is dealing with deafness of this type must take the history carefully in order to decide whether the deafness came on suddenly. Fowler, Sr.,[1] stated that some persons have been known by their friends and relatives to be totally deaf in one ear for years but have not been aware of it themselves. The history may reveal that they suddenly noticed their deafness when the normal ear was buried in a pillow, and they could not hear a speaker standing a short distance away. Deafness also may and does actually come on like "a bolt from the sky"; sometimes the patient wakes up with it. In cases of sudden onset tinnitus is always severe and often distresses the patient more than the deafness.

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* Submitted as Candidate's Thesis to American Laryngological, Rhinological and Otological Society, Inc., 1955.

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REVIEW OF LITERATURE.

Sudden deafness of obscure origin has received rather scant attention in the literature. Prosper Ménière[2] apparently was the first to report such a case and elaborate on the possible etiology and pathology. Kobrak[3] reported eight cases in 1922 but actually only one of his patients had sudden deafness with no obvious cause. De Kleyn,[4] in 1944, described "complete or partial loss of function of the octavus system in apparently normal persons." In the summary he mentioned 21 cases; actually he had 24 cases, in two of which the involvement was bilateral. He considered vascular disorders as the underlying cause. In some cases a more or less complete recovery was obtained. Rasmussen,[5] in 1949, reported 18 cases of sudden deafness of unknown etiology, in 15 of which the deafness was combined with vestibular disturbances. In discussing the etiology, he stated that in three cases the cause might have been thrombosis of the internal auditory artery or one of its branches, or hemorrhage into the labyrinth due to rupture of a vessel. In a fourth case vascular spasm was considered a possibility. In the remaining cases the cause was supposed to be neuritis of the VIIIth cranial nerve.

In March, 1950, Lindsay and Zuidema[6] gave an excellent review of 16 cases of inner-ear deafness of sudden onset. The first four cases were types in which the aural condition was associated with systemic disease. In the 12 cases in which the deafness was associated with a varying degree of vestibular disturbance the cause was uncertain. Lindsay and Zuidema further stated that the same etiologic factors cannot be assumed in all 12 cases, but that the similarity of the clinical picture suggests the same type of cause. A vascular accident in the labyrinth has commonly been the explanation, but the high incidence among adults less than 30 years of age argues against this cause. They also considered the possibility of vasomotor disturbances of the inner ear and recommended that toxic involvement of the labryinth be given serious consideration, since it is a well-known occurrence in infections of various types.

In December, 1950, Fowler[7] reported 26 cases of sudden deafness, and he attempted to include everything which might have some bearing on the etiology. About 77 per cent of his patients were less than 40 years of age. Fowler's most important finding was a history of emotional episodes before or immediately preceding the sudden deafness. The patients, however, rarely seemed to blame the emotional condition for the deafness. Eighteen of the 26 patients apparently suffered from psychosomatic disorders. Fowler stated that such episodes are known to affect the neurovascular mechanisms through vascular contraction, sludging and congestion ending in anoxia and leakage from capillaries and local metabolic deficiencies. Five of Fowler's patients examined for sludging showed it distinctly. He stated further that all or any one of these factors may be and frequently are the cause of sudden deafness.

In 1950 also, Opheim[7] reported five cases of sudden deafness at the Eleventh Northern Oto-Laryngological Congress. He was much impressed by Mygind's[8] idea, presented at a meeting of the Danish Oto-Laryngological Society, that the loss of hearing might perhaps be brought on by an acute rise of pressure in the labyrinth. Opheim stated that this hypothesis opens a road to a rational intervention against this condition, which otherwise has such an unfavorable prognosis. Fenestration operations were carried out for their decompressive effect on three of his patients. At operation an attempt was made to leave the ossicular chain intact. In all three cases the hearing improved some but not to a serviceable level. On puncturing the labyrinth, the fluid was found to be under pressure in one patient, but not in the other two. Opheim stipulated the following conditions for operation: *1.* The loss of hearing must be complete to the extent that such a condition is recognizable, the function of the other ear being normal. *2.* The patient must be available for treatment at such an early time in the course of disease, that it may reasonably be supposed that the damage to the sensory apparatus has not become irreparable.

Mygind,[9] in discussing Opheim's paper, reported the case of a colleague with sudden deafness. When this patient was admitted to the hospital, his hearing had returned to some extent. Because of this, treatment for Ménière's disease was started, and the hearing became normal. Mygind stated that if the hearing does not return in such cases he would give dextran (macrodex), a polysaccharide with especially large molecules which cannot pass through the capillary walls.

In 1952, Moulonguet and Bouche[10] reported one case of sudden deafness. The patient, a young doctor, became suddenly deaf in the right ear a few hours after receiving an injection of "orthobiotic" serum. The patient was examined on the fourth day after the injection. The audiogram showed marked loss of hearing in the speech range (65 db). Bone conduction was poorer than air conduction and Fowler's test showed recruitment for the frequencies 512 and 1,024. Moulonguet and Bouche considered that this condition was caused by a vascular disturbance or more precisely an arteriolar spasm. The patient was slowly given an intravenous injection of sodium nicotinate; the usual marked peripheral congestion followed. Three hours later hearing had returned to normal. Administration of sodium nicotinate was continued by the intramuscular route for a week, and the hearing has since remained normal. They commented about as follows: *1.* Because of recruitment, the lesion seemed to be localized to the cochlea. *2.* The spectacular result of a peripheral vasodilating agent confirmed the hypothesis of arteriolar spasm.

MATERIAL STUDIED AND OBJECTIVE OF STUDY.

In the five years from 1949 to 1953, inclusive, 178 patients with sudden deafness of obscure cause were seen at the Mayo Clinic. These cases were studied carefully to determine, if possible, the cause of the deafness. The objective of this study was to determine if possible the underlying cause of the sudden deafness and the result of treatment, if any, with the view of aiding physicians to arrive at the diagnosis.

ETIOLOGY, PATHOGENESIS AND PATHOLOGY.

From the review of the literature, it is evident that generally the etiology of sudden deafness of the obscure type under consideration is unknown, but several possible causative factors were suggested. These briefly are *1.* thrombosis, vascular spasm or hemorrhage; *2.* neuritis of the VIIIth cranial nerve; *3.* systemic disease; *4.* toxic involvement of the labyrinth with infections elsewhere; *5.* emotional episodes; *6.* acute rise in pressure in the labyrinth; 7. injection of an "orthobiotic" serum and 8. Ménière's disease.

Factual information about the pathologic process underlying the loss of function has been impossible to obtain because deafness is seldom or never associated with events that kill the patient.

In 89 of the 178 cases studied deafness seemed to be of vascular origin; in 56 it seemed to be due to Ménière's disease (endolymphatic hydrops); in 17 the cause could not be determined; in 15 it seemed due to miscellaneous causes; and in one case, to hysteria or malingering. The clinical features which led to this decision and any available data concerning diagnosis, differential diagnosis, pathology and pathogenesis will be considered when the types are discussed.

SUDDEN DEAFNESS OF POSSIBLE VASCULAR ORIGIN.

The reasoning behind a diagnosis of vascular accident in a case of deafness of sudden onset is about as follows: The vascular supply of the labyrinth is such that a thrombus, vascular hemorrhage or vasospasm might affect the entire auditory sense organ or only part of it and produce sudden deafness. If the sudden deafness is bilateral, a hydrops type of disorder must be considered because it is unlikely that vascular accidents should occur in both ears at the same time. A history of one attack of sudden deafness with tinnitus in one ear followed in minutes or hours by a dizzy spell, which persists for a few hours to a month but with persistent loss of hearing, is suggestive of a vascular accident. The deafness may often be total and permanent.

Pathologic Hypotheses: Mygind[9] expressed the view that many patients who become deaf suddenly must have hemorrhage into the modiolus. Vestibular symptoms occur only later when bleeding or edema spreads to the nervous system of the labyrinth. The general term "vascular accident" was used by Lindsay and Zuidema.[6] Fowler, Sr.,[1] said that this term was "unscientific;" however, I have used it in this paper, because it implies vascular damage but does not specify the exact type.

Kobrak[7] stated that hemorrhage always should be suspected whenever deafness develops suddenly, even if there are no signs of hemorrhagic diathesis. If the damage to the inner ear seems to be reversible, transient vasospasm may be an important factor. Occasionally patients have had one severe dizzy spell without any deafness. Subsequent caloric tests often, but not always, reveal a nonfunctioning equilibratory labyrinth on one side. It seems reasonable to assume that in these cases a vascular accident may have involved the vestibular branch of the internal auditory artery only.

A look at the cases shows other features which are compatible with a diagnosis of vascular accident.

TABLE I.

AGE DISTRIBUTION OF PATIENTS WITH DEAFNESS OF VASCULAR ORIGIN AND OF MENIERE'S DISEASE

	Vascular Origin		Ménière's Disease	
	No.	Per Cent	No.	Per Cent
Males	43	48.3	26	46.4
Females	46	51.7	30	53.6
Age, Years				
20-29	9	10.1	2	3.6
30-39	18	20.2	15	26.8
40-49	18	20.2	20	35.7
50-59	21	23.7	12	21.4
60-69	18	20.2	7	12.5
70-79	5	5.6		
TOTAL	89	100.0	56	100.0
Average age, years	49.0		46.3	
Youngest, years	22		20	
Oldest, years	73		66	

Age and Sex: Of the 89 patients who had sudden deafness which seemed to be due to a vascular accident, 43 were males and 46 were females (see Table I). A significant number (27) were from 20 to 39 years old; however, the majority of patients were evenly spread over the older age groups. This age distribution compares fairly well with the age incidence of coronary occlusion. Parker and associates[11] found that 85 per cent of their 3,400 patients with angina pectoris were from 40 to 69 years of age; 64.1 per cent of the 89 patients with sudden deafness of possibly vascular origin in my group were in the same age period. There was a significant discrepancy, however, as only 3.6 per cent of the patients with angina pectors were 20 to 39 years of age, but 30 3 per cent of the 89 patients in this series were in this age group It is difficult to explain this discrepancy, but one should not forget that branches of the internal auditory artery are end-arteries, so that even minor vascular changes might cause early symptoms

General Examinations and Pertinent Findings Which Bear on the Cause of the Deafness The average time from the onset of deafness until the patient was examined at the clinic was four months Sixty-one (69 per cent) of the 89 patients had general examinations conducted by an internist and 42 patients had a complete neurologic evaluation at the clinic

Twenty patients had different degrees of hypertension, arteriosclerosis, or both, with hypertensive changes in the ocular fundi A few were checked for blood sludging as suggested by Fowler, Jr ,[1] but this was abandoned because of variable findings Rucker[14] expressed the opinion that these might be caused by the heat of the slit lamp Only two patients had blood dyscrasias one had polycythemia vera, the other, chronic lymphatic leukemia Sudden deafness followed a severe transfusion reaction in one case The patient had been given Rh positive blood, and he was Rh negative

Hearing The deafness of all 89 patients seemed to come on without any apparent cause Ten patients woke up with severe roaring and deafness in one ear Three patients became deaf while using the phone Allergic factors or emotional problems did not seem to play a significant part

All 89 patients except those who were totally deaf had audiograms, speech-reception tests and loudness balance tests In 32 patients (36 0 per cent) the deafness was total in one ear Right and left ears were involved with about equal frequency Two patients had bilateral involvement, in these two the deafness was total and permanent The intervals between onset of deafness in the first ear and the second ear were three weeks and one year respectively The caloric tests revealed nonfunctioning equilibratory labyrinths on both sides Both patients had compensated well, and were able to move about well, except in darkness

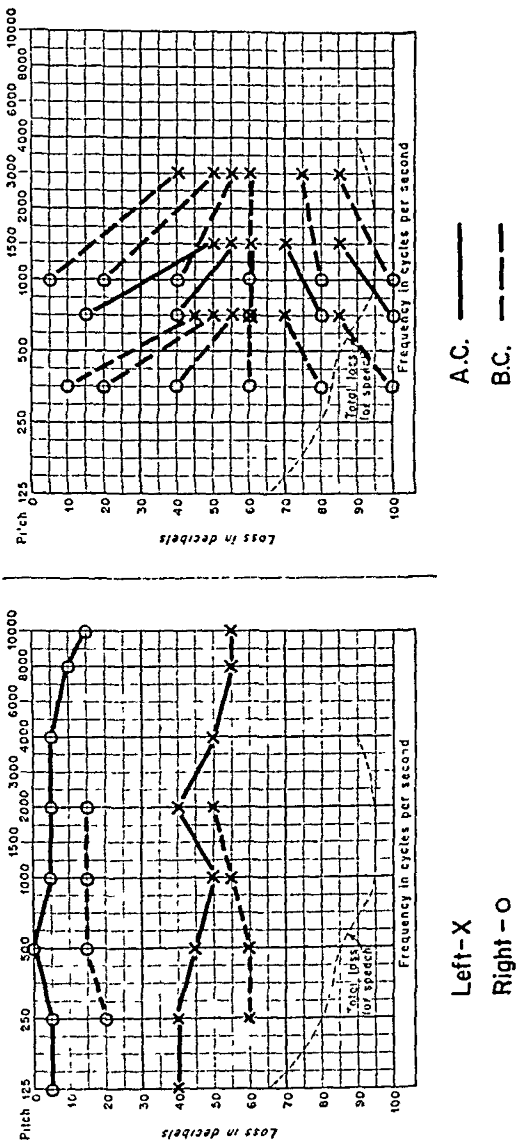


Fig. 1. Case 1. Audiograms and result of loudness-balance test in typical patient with deafness of vascular origin which involved the entire tonal range.

The audiograms of the entire group of 89 patients revealed great variation in the deafness, but it was always of the end-organ type. This diagnosis was confirmed by loudness-balance and speech-reception tests. Thirty-two patients (36.0 per cent) had flat curves with deafness of varying degree. Case 1 is a typical example.

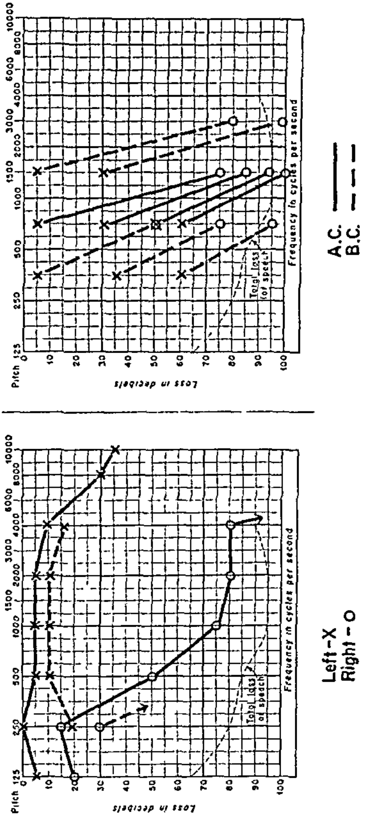


Fig. 2. Audiograms and result of binaural loudness-balance test of patient with deafness of vascular origin involving mainly the hearing of high tones

Case 1: The audiogram and result of loudness-balance test for a woman 62 years old revealed a moderate end-organ type of deafness with marked recruitment in the left ear over the right (see Fig. 1). Reexamination a year later did not reveal any change in hearing; however, recruitment was much less marked, and there was no evidence of overrecruitment. The patient felt that she could hear better because there was less confusion.

Seventeen patients (19.1 per cent) had mainly loss of hear-

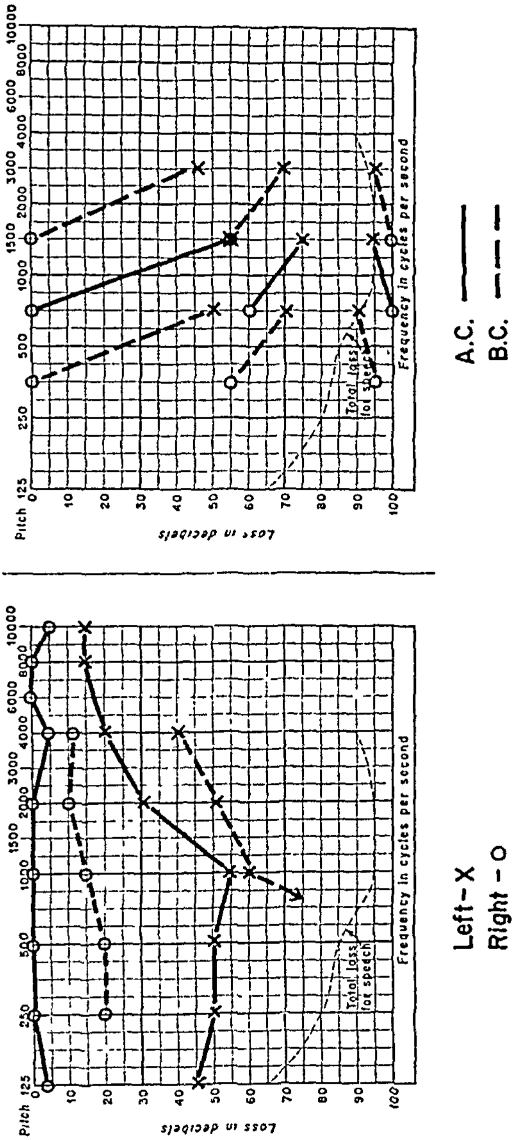


Fig. 3. Audiograms and result of loudness-balance test of a patient with vascular type of deafness involving low tones.

ing for high tones, but the degree of this loss varied. The audiogram and the result of recruitment test of a representative case are shown in Fig. 2. The patient, a woman 59 years old, had suddenly lost her hearing, while playing with her grandchildren. Re-examination two months later revealed no change in hearing. The caloric test revealed normal responses.

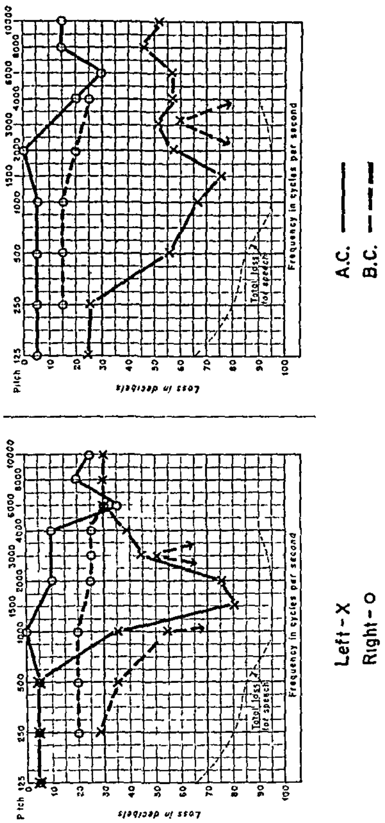


Fig 4 Case 2 Audiograms of a patient whose sudden deafness started as an island type

In four patients (4.5 per cent) the low tones were mainly involved. The audiogram and the result of recruitment test of a man, 35 years of age, who had involvement of the low tones, are shown in Fig. 3. Sudden roaring tinnitus had developed in the left ear with deafness but no dizziness five months prior to examination. Hearing has remained unchanged since.

In four patients (4.5 per cent) the deafness was of an island type.

Case 2: One such patient, a woman 48 years old, woke up one morning with severe ringing and deafness in the left ear. She was seen the following morning. Results of the ear, nose and throat examination were negative. The left Eustachian tube was patent. The audiogram (see Fig. 4) revealed a marked dip involving mainly the range of 1,500 to 2,000 frequencies. Treatment was started immediately. She was instructed to take a form of nicotinic acid (roniacol) in tablet form, 100 mg. before breakfast and 100 mg. before supper. Four days later the hearing was definitely worse. Deafness had spread over the neighboring frequencies. When she was examined again one month later, her hearing was essentially normal. The patient was re-examined one and two years later, and the hearing remained normal.

Because of the single attack only, this deafness was felt to be primarily vascular in origin.

Dizziness: In 41 of the 89 patients in this group (46.1 per cent) deafness and tinnitus were the only symptoms. In 48 patients (53.9 per cent), the loss of hearing was associated with varying degrees of dizziness, nausea and vomiting. Usually the vertigo started a few minutes to 12 hours after the onset of the deafness. This could be explained by retrograde thrombosis causing complete or partial blockage of the vestibular branch of the internal auditory artery. More females had dizziness associated with deafness than males. All 48 patients who gave a history of unsteadiness or dizziness at the onset of the deafness underwent caloric tests. About 70 per cent had varying degrees of hypoactivity of the equlibratory labyrinth. Fifteen of the 32 patients with total deafness in one ear and dizziness gave no response to caloric stimulation on the side of the deafness. In the other cases, however, there seemed to be no definite relationship between the degree of deafness and the result of the caloric test. Some of the patients were tested who had no history of dizziness. All had normal responses on both sides, as one would expect. As has been stressed by Lindsay and Zuidema,[6] the sudden onset, the single attack only, and the usually permanent loss of function are sufficient to differentiate the condition from Ménière's disease or hydrops of the labyrinth. In two of our patients the vascular involvement was more extensive. Details of one are given in Case 3.

Case 3: The patient, a man aged 53 years, stated that two months prior to examination sudden total deafness in the left ear, slight dizziness and incomplete facial palsy on the left side developed. Examination revealed total deafness, facial weakness and diminished corneal reflex, all on the left side. Complete general and neurologic examination otherwise gave negative results.

This patient probably had a vascular accident involving parts of the pons and brain stem, and possibly the posterior inferior cerebellar artery.

Because of all the facts mentioned in this section the pathologic disturbance occurring in the 89 patients classified as having deafness due to vascular accident seems different from that in the next group to be discussed, in which the sudden deafness seemed to be caused by Ménière's disease or hydrops of the labyrinth.

SUDDEN DEAFNESS APPARENTLY DUE TO MENIERE'S DISEASE (ENDOLYMPHATIC HYDROPS).

Etiology, Pathogenesis and Pathology of Sudden Deafness Thought to Be Ménière's Disease: I was unable to find a report of detailed microscopic examination of a temporal bone from a patient with sudden deafness; however, it has been reported that sudden deafness often represents the onset of Meniere's disease. For this reason certain studies on Meniere's disease will be considered here.

The first microscopic observation of the temporal bones of persons known to have had severe attacks of vertigo, deafness and tinnitus during life was reported by Hallpike and Cairns[14] in 1938. They found hydrops of the labyrinth, and it was reasonable to conclude that the effect of this hydrops on the cochlear duct, utricle and saccule was responsible for the symptoms.

Many studies since then have confirmed the histopathologic incidence of an endolymphatic distention of the cochlear duct without inflammatory reaction. As a result of these observations the terms "endolymphatic hydrops," "hydrops of the labyrinth" or "labyrinthine dropsy" are now commonly used to designate this disease.

Although the histopathologic reports are increasing, certain fundamental questions concerning the disease have not been answered. The exact etiology and pathogenesis have not been clearly determined.

Hilger[15] attempted to show that the vital intralabyrinthine structures depend for integrity of function and structure on end-arteries, which do not have collateral circulation. He suggested that a disturbance in the vascular function common to all end circulation can cause vasospastic changes in the labyrinth. These may result in primary neurovascular ischemia and endolymphatic hypertension, which bring about this syndrome. Williams[16] suggested the hypothesis that Ménière's disease is an instance of autonomic imbalance, specifically a cholinergic preponderance or overactivity. He theorized further that Ménière's disease is a form of intrinsic allergy involving the capillary loops of the stria vascularis and that the endolymphatic distention is a localized extracellular edema produced by the spastic-atonic state of the capillaries.

Day[17] stated that in an occasional case a specific protein allergen may prove to be the causative factor and reported observations in several cases in which the symptoms could be abolished by removal of the offending allergens only to recur with their reintroduction into the patient's environment. Day also presented evidence that in labyrinthine hydrops the cochlea is primarily affected and the vestibular disturbance is due to secondary or overflow reaction of the vestibular mechanism.

More recently Lempert and his co-workers[18] have reported on histologic changes in the inner lining of the membranous walls in the endolymphatic external semicircular canals of six patients with Ménière's disease who underwent partial labyrinthectomy for the arrest of persistent dizzy spells. They found that this inner membranous lining of all specimens was studded with vesiculated epithelial excrescences which suggested collections of epithelial fluid. They postulated that an attack of Ménière's disease is due to the rupture of one or more vesicles with release of toxic fluid into the lumen of the endolymphatic membranous labyrinth. They also postulated that Ménière's disease may be primarily a disease of the vestibular labyrinth and that the pathologic changes in the cochlea and the symptoms are the sequelae of vestibular disease. They concluded that the cumulative loss of hearing during the course of the disease is due to progressive damage of the organ of

Corti by the repeated contamination of the endolymph by this vesicular toxic fluid.

Shimamoto and co-workers[19] in 1954 reported some interesting experiments on 160 guinea pigs. They replaced the endolymph with various chemical substances, especially solutions of potassium chloride in various concentrations. They suggested that some disturbance of the secretion or absorption of the potassium of the endolymph may exist in Ménière's disease and if the concentration of potassium could be changed, especially by being increased unequally in both endolymphs, an excitatory syndrome of the vestibular organs would be established just as in Ménière's disease. They considered that it was not so unnatural to speculate that the cochlea might also be affected by an unusually high concentration of potassium in the endolymphatic system.

General Observations on the Group: In the five years under consideration a total of 1,270 patients were seen at the Mayo Clinic and given a diagnosis of true Meniere's disease. The Ménière's disease of 56 patients (4.4 per cent) started with sudden deafness. This figure may in reality be higher since so many of the patients with Ménière's disease are so distressed by the severe tinnitus and dizziness that they do not give the deafness much thought.

Repeated examinations are required in order to permit a correct diagnosis. The history is extremely important. As has been stressed often by Fowler, Jr., and Zeckel,[20,21] emotional factors played an important role in precipitating an attack. This psychosomatic influence seemed, however, to be much more at play *after* the disease had started, and seemed to be a participating factor only in causing repeated attacks. The *sudden deafness usually came out of a clear sky and was or* was not associated with dizziness. There were remissions and exacerbations of deafness with usual progression of deafness over the years, no matter what type of therapy was used.

Age Distribution and Time of Examination: The age distribution is shown in Table I. Forty-seven (83.9 per cent) of the 56 patients first had their trouble between the ages of 30 and 59 years and only seven patients became deaf after the age of 60 years. The mean age at onset was 46.3 years.

The average time from the onset of deafness until the patient was examined at the clinic was eight months, four months longer than for the first group of patients.

Deafness: As in the "vascular-accident" group all patients were checked with audiograms, speech-reception tests, and loudness-balance tests except where the deafness was total. Here again the deafness varied but was always of the end-organ type. In six patients the deafness was total, or nearly so; in one of these patients the total deafness was bilateral.

Case 4: Another one of these six patients, a man 33 years of age, had suddenly lost his hearing in the left ear at work two weeks prior to examination. Severe dizziness, nausea and vomiting started the following day. Initially, he also had some ringing in the right ear. Dizziness persisted. Ear, nose and throat examination gave negative results; however, the audiogram revealed almost total deafness in the left ear and a moderate amount of end-organ type of deafness in the right. The patient was treated with mono-ethanolamine nicotinate (nicamin), ammonium chloride and low-salt diet for two months, then booster courses were given to ward off symptoms. Audiograms made about one month later revealed no change in hearing on the left side but definite improvement on the right side. The caloric test revealed a nonfunctioning equilibratory labyrinth on the left side and normal responses on the right. On re-examination a year later hearing was unchanged in the left ear, but had improved further in the right.

A surprisingly large number of patients (19) had bilateral deafness. In 13 of these 19 the deafness came on suddenly in both ears and at the same time, and in 12 it was associated with vertigo, nausea and vomiting. In three patients there was an interval of from six hours to a month between the sudden onset of deafness in the first and the second ear. In one patient the interval was nine years. In two patients the onset of deafness was gradual in one ear and sudden in the other. In most of these patients hearing fluctuated after the initial attack.

Generally speaking, the more severe the deafness was at onset, the more likely there was to be bilateral involvement and the less chance for benefit from treatment. Involvement of the entire tone range also seemed to lessen the chance of benefit from treatment. Seventeen patients (39.0 per cent) had involvement of the entire range of tones. Case 5 is a typical example.

Case 5 The patient, a man 50 years of age, became deaf suddenly five months before he was examined at this clinic, and in the intervening months before admission had had several severe dizzy spells At the clinic the regular Ménière's disease regimen was instituted with salt-poor diet and doses of nicotinate (nicamin) and ammonium chloride When the patient was re-examined three months later he stated that he felt better, had had no further dizzy spells and felt his hearing had improved The audiogram and loudness balance test revealed, however, that the hearing was unchanged. The loudness balance test still showed recruitment but it was much less marked. This finding means less distortion of speech.

None of the 10 patients (61 per cent) with involvement of the entire tonal range who were re-examined had any real improvement of hearing, as indicated by audiograms and speech-reception tests; however, slight variations in hearing from day to day were noted.

The patients who had sudden deafness involving only part of the tonal scale would have fluctuation in their ability to hear, and 14 (about 40 per cent) ended with normal or nearly normal hearing with or without treatment. Of these, seven patients had mainly a loss of high tones; five patients had mainly loss of low tones and only two had deafness which was confined to the speech range. One patient (Case 6) is of special interest because she had lost ability to hear high and low tones in the same ear, but these changes came on at different times.

Case 6 The patient, a woman 37 years old, lost her hearing in the right ear suddenly while she had both hands in cold water, a roaring tinnitus and "numb feeling" occurred in the right side of the occiput. Two days later a severe dizzy spell associated with nausea and vomiting developed The audiogram taken five days later showed a moderate loss of hearing for high tones of end organ type with marked recruitment Caloric tests revealed normal responses on both sides She was treated with mono ethanolamine nicotinate (nicamin), ammonium chloride and salt-poor diet Within a week the hearing returned to normal About a week later, she again lost the hearing in the same ear (right) suddenly. This episode was also accompanied by dizziness and nausea This time the low tones were also involved.

Audiograms and a loudness balance test were made six days later and an audiogram two months later. By that time her hearing was essentially normal.

Dizziness: The dizziness varied from a transient unsteady feeling to a violent vertiginous attack associated with nausea and vomiting. Usually the more severe the associated dizzy spell, the more it would show up in the result of the caloric test.

Only 16 patients (28.6 per cent) did not give a history of any dizziness. The remaining 40 (71.4 per cent) gave a history of dizziness of varying intensity. In most of them the dizziness came on either at the same time as the deafness or a few hours to two days later. In five patients the vertigo was the initial symptom.

In the six patients who had total or nearly total deafness, there generally was marked depression of caloric function. In 36 of the 40 patients who had dizziness associated with deafness, the equilibratory labyrinth was hypoactive or nonfunctioning. In four of the 40 patients responses were either within normal limits or hyperactive.

The carefully elicited general history did not give any definite clues as to what might have precipitated the hydrops. There did not seem to be any definite allergic relationship and only nine (16.1 per cent) had hypertension, arteriosclerosis or both with sclerosis of the retinal arteries of varying degree. My colleagues and I were unable to obtain a definite history of emotional upsets at the onset; however, they seemed to be important in precipitating subsequent dizzy spells with or without further deterioration of the hearing.

DEAFNESS OF UNDETERMINED CAUSE: HYDROPS OR VASCULAR ACCIDENT.

In 17 cases the diagnosis could not be made definitely. Most of these patients were seen only once or were seen too recently to permit evaluation of the possible cause.

SUDDEN DEAFNESS OF MISCELLANEOUS ORIGIN.

Deafness of sudden onset, sometimes associated with vertigo, is known clinically to occur on a toxic basis as a complication of some infectious diseases, which are mainly virus in type. Lindsay and Zuidema[6] stated that there is no constant pattern with regard to the proportion of hearing loss that is due to vestibular dysfunction as a result of a toxic process.

In most of the 15 patients in this group the deafness was considered as toxic in origin. The youngest patient was six, the oldest 74 years of age. About half of the patients were between 40 and 49 years of age. All 15 patients in this group had complete general and neurologic examinations, audiograms, loudness-balance tests, speech-reception tests and caloric tests, if deafness was associated with dizziness.

Deafness: As in the first two groups the deafness varied from total bilateral to moderate unilateral deafness. The deafness was of an end-organ type with evidence of moderate to marked recruitment in all except two patients. One of these two who had no recruitment had multiple sclerosis, and the *other had a tumor of the cerebellopontine angle.*

TABLE II.

SUDDEN DEAFNESS OF MISCELLANEOUS ORIGIN

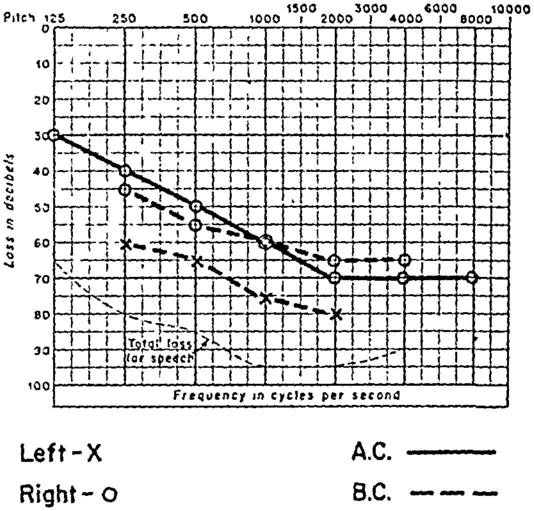
Intimate Relationship or Cause of Deafness	Patients
After operation and general anesthesia	2
Heavy dosage of aspirin for arthritis	2
Syphilis	2
Peptic ulcer, abscess and antibiotics	1
Severe acute upper respiratory infections	2
Large dosage of alcohol and bromoquinine	1
Herpes zoster	1
Booster shot for whooping cough (case 7)	1
Multiple sclerosis (case 8)	1
Cogan's syndrome (case 9)	1
Cerebellopontine-angle tumor (case 10)	1
TOTAL	15

As might be suspected, in eight of 15 patients (53.3 per cent), the sudden deafness was bilateral, and in three the deafness was total.

The deafness of only five patients was associated with dizziness. All five had bilateral loss of hearing. This observation tends to confirm the statement that the equilibratory apparatus is much more resistant to toxic influences than the cochlea. After the initial loss, the hearing remained down in 13 patients. In the other two, congenital syphilis was thought to be the cause of the toxic effect. The hearing of one of these two patients fluctuated rather markedly after antisyphilitic treatment, but two months after the treatment was started the hearing remained down. In the other patient, in spite of initial fluctuation in hearing, the hearing in the left ear disappeared entirely.

he cause of the toxic effect, elicited from the history, was ed and difficult to nail down exactly. The diseases or con- ons which were active or present at the onset of the sudden ness in these 15 cases are presented in Table II.

he last four cases (Cases 7 to 10) referred to in Table II unusual and deserve further comment.



g. 5. Case 7. Audiogram of patient with sudden deafness from whoop- cough-booster injection.

ster Injections for Whooping Cough.

se 7: The patient, a girl aged six years, was first seen Aug. 4, 1951. mother gave the following history: Her sister had had whooping h and the local physician advised booster shots for the patient. On way home from the doctor's office, a 25-mile drive, the patient said ears felt "funny." As soon as they got home the girl said she could hear a thing. The patient vomited at first but did not stagger; her onality changed and temper tantrums and overactivity developed. The nt was seen two and half months after the onset of deafness. lts of the ear, nose and throat examination were essentially negative. first attempt to check the hearing with audiograms did not seem rate. The testing was done again a few days later without the nts being present (see Fig. 5). The child then was much more erative.

hysterical reaction had to be ruled out. The patient could not ate electrodes for galvanic skin tests. Nothing definite was found omplete neuropsychiatric checkup except oversolicitous parents. Since onset of the deafness there had been no definite fluctuation in hear- and the child had already become an efficient lip reader. Active

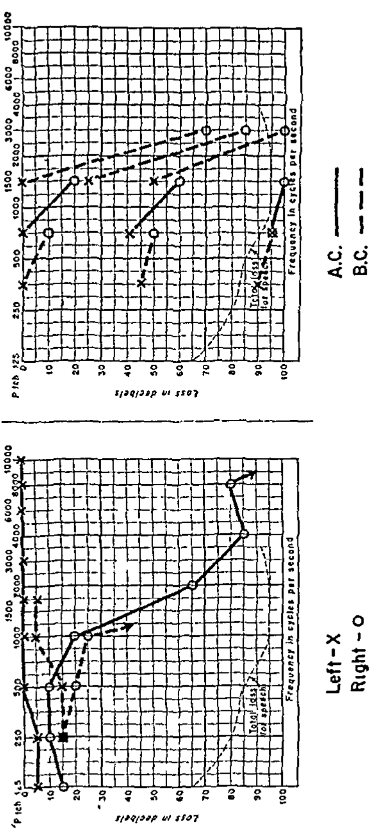


Fig 6 Case 8 Audiograms and recruitment test of patient with multiple sclerosis

training at home was suggested, and the parents were advised to contact the John Tracy Institute for home training programs The caloric test was not done; however, the patient had not experienced any dizziness or had any trouble in walking and playing The final opinion was that the sudden deafness was probably caused by an anaphylactic phenomenon either involving the nerve or cochlea Because of poor speech reception on the right side, indicating distortion, the cochlea itself probably was affected.

I was unable to find a similar case in the literature. Sudden deafness following tetanus antitoxin, however, has been reported. An excellent description and bibliography were given by Berger and Sachs[22] in 1953. Their case was the third reported in the literature. In the three cases reported in the literature the deafness came on about a week after the injection during an eruption of serum sickness (generalized urticaria).

Sudden edema of the labyrinth is probably responsible for the deafness due to an anaphylactic reaction; however, the knowledge about deafness caused by allergy, serum sickness and anaphylaxis is not complete enough to permit a statement of the entire true picture of the pathology.

Multiple Sclerosis.

Case 8: Two years before the patient, a woman aged 27 years, was first seen, a roaring sound had developed suddenly in the right ear, which had felt completely "blocked up." The roaring lasted two weeks and then subsided, but deafness persisted. No dizziness was noted. The doctor in the home locality gave her histamine, but it did not help. The hearing never seemed to fluctuate. For about two months she had spells of slurring speech, transient numbness of the left side of the body and some "dragging" of the left leg. Examination of the ears, nose and throat gave essentially negative results. The Eustachian tubes appeared to be patent. The audiogram revealed marked deafness for high tones on the right and binaural loudness-balance test showed significantly no recruitment (see Fig. 6).

The caloric responses were normal on both sides. The significant ocular findings were: *1.* spontaneous vertical nystagmus on looking up and *2.* sheathing of the retinal veins, which according to Rucker[23] is found in about 10 per cent of patients with multiple sclerosis. General examination was noncontributory; however, the neurologic examination revealed some numbness and weakness of the left leg. A diagnosis was made of multiple sclerosis. Because the deafness was of a nerve type with no evidence of recruitment, it seemed to be caused by the same disease process. It seemed probable that the disease attacked the auditory nerve itself, corresponding to optic neuritis, which is commonly found in multiple sclerosis.

In 1888 Hess[24] reported auditory findings in a case of multiple sclerosis which he had observed for more than eight years. Sudden bilateral deafness became permanent in the right ear, while the hearing in the left ear returned gradually after 24 hours. Sudden loss of hearing associated with multiple sclerosis is rare. Apparently none of the 92 patients studied by Von Leden and Horton[25] experienced sudden deafness. These investigators found, however, that defects in the auditory field, comparable to defects in the perimetric field, occur fairly frequently.

Bentzen and associates[26] stated that in most cases of multiple sclerosis in which impairment of hearing has occurred, it has been transitory, being mentioned usually as a link in acute pontobulbar outbursts. A continuous analysis of 64 patients revealed a considerably higher auditory activity of the disease than is usually reported. A fifth of the patients had impairment of tone perception. The acoustic disturbances (including tinnitus) caused marked subjective discomfort in only a few.

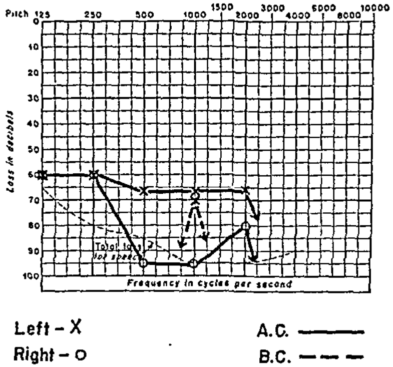


Fig. 7. Case 9. Audiogram of patient with Cogan's syndrome.

Phillips[27] stated that symptoms referable to the ears were outstanding events at the onset of multiple sclerosis in two per cent of cases and the most frequent such symptom is vertigo. Phillips expressed the opinion that the deafness is caused by actual involvement of either the auditory nerve or the cortex of the brain in the auditory radiation. Krohn,[28] of the University of Oslo, Norway, told me that he had never seen a case in which sudden deafness was an outstanding symptom.

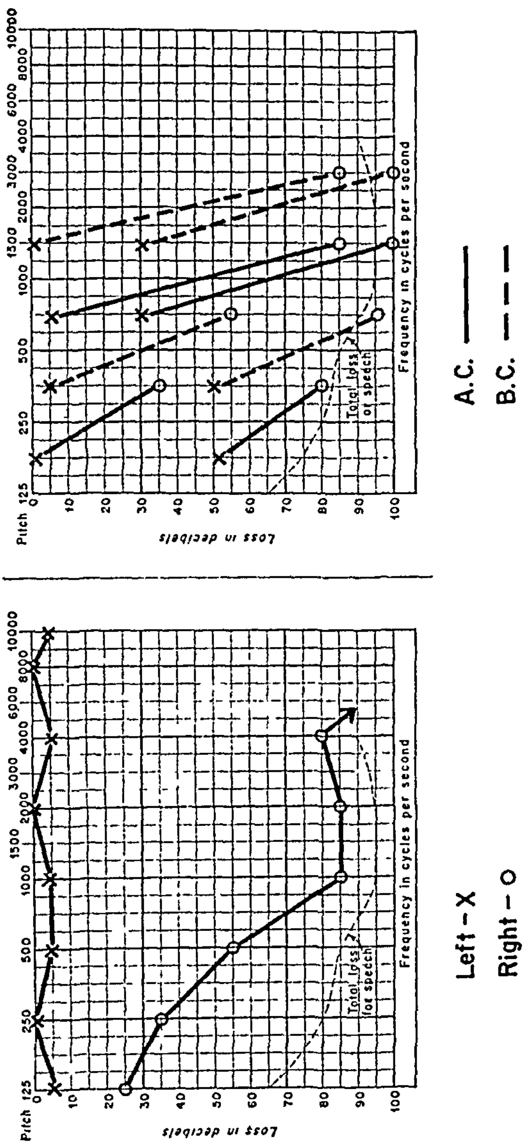


Fig. 8. Case 10. Audiograms and result of loudness-balance test of patient whose deafness was due to a tumor of the cerebellopontine angle.

Cogan's Syndrome.

Case 9: The patient was a 27-year-old man who, five months before the examination, was dizzy one morning on arising. He also felt nauseated but did not vomit. Three days later sudden bilateral tinnitus and deafness developed. He said he could not hear; however, hearing returned gradually within the next few days, but deafness returned suddenly three months later. Dizziness disappeared, but now the deafness remained At about the same time the eyes became reddened, and the redness persisted

Except for profound deafness the ear, nose and throat examination gave negative results (see Fig 7). Caloric tests revealed nonfunctioning equilibratory labyrinths on both sides. Examination of the eyes revealed corneal opacities of a vascular type, presumably in the interstitial layers of the cornea. Results of the general examination and of flocculation tests for syphilis were negative

This was evidently the syndrome of nonsyphilitic interstitial keratitis with vestibular auditory symptoms first described by Cogan[29] in 1945. The patient was treated with aureomycin and histamine for a month. Reexamination and audiogram then showed no change

Cogan's syndrome usually occurs in young adults and is often of sudden onset, either with ocular or aural symptoms. According to Olner and associates,[30] symptoms from eyes and ears may occur within a few hours of each other, or at any interval up to five months. In the majority of cases the interval is between one and two weeks. Lindsay and Zuidema[6] reported three cases of this type with sudden loss of hearing. In all their cases, as in mine, the deafness was bilateral. The etiology is not known; however, the inflammatory nature of the disease seems definite.

Cerebellopontine-Angle Tumor.

Case 10 The patient, a doctor's wife, 46 years of age, first was seen in 1947 for a pelvic operation She returned in March, 1953, for a routine examination and for a check of hearing because of deafness on the right, which had come on suddenly two years before while she was walking over a frozen lake. Roaring tinnitus was associated with the deafness at first but subsided in a couple of weeks The deafness, however, became gradually more severe. She would not have consulted the clinic for this if she had not accompanied her husband.

Results of ear, nose and throat examination were essentially negative The audiogram (see Fig 8) revealed a marked nerve-type deafness on the right, and the binaural loudness balance test showed conspicuous absence of recruitment The caloric test revealed a nonfunctioning equilibratory labyrinth on the right. The corneal reflexes were normal on both sides, however, a fine rotatory nystagmus was present on lateral gaze to the left. The quick component appeared to be clockwise.

Except for these findings the neurologic examination was entirely negative, and there was no evidence of choked disks Roentgenograms of the head, including Stenver's views, were negative The only other significant finding was increased protein (190 mg per 100 cc) in the spinal fluid. General examination showed mild hypertension

On the strength of the rather meager findings the right cerebellopontine angle was explored and a neurofibroma 2 5 cm in diameter was removed in pieces Incomplete facial palsy developed on the right side, but otherwise the patient's postoperative course was uneventful

Sudden deafness associated with cerebellopontine-angle tumors is rare. Cushing[31] reported on the sudden onset of deafness of the left ear associated with violent vertigo. Most patients with tumors of the cerebellopontine angle do not have real vertigo. Both Cushing's and my cases probably represent a vascular accident involving the blood supply to the nerve which was brought on by encroachment of the tumor.

PSYCHOGENIC DEAFNESS (EMOTIONAL DEAFNESS).

I tried to exclude the possible influence of psychogenic deafness on results by keeping in mind the differential diagnosis between this type and other types of perceptive deafness. Psychogenic deafness is caused by a combination of psychic and organic factors; the latter may be any kind of hearing loss. There are certain clues for detection and Doerfler[32] listed the following: *1.* sudden onset of bilateral severe hearing loss, perceptive in nature; *2.* saucer-shaped curve in audiogram (50 to 90 decibels) in 80 per cent of his cases; *3.* unexplainable discrepancy between audiometric results and tests for hearing of speech, especially when the hearing for speech is better than that for pure tones; *4.* surprising response to therapy out of proportion to what may reasonably be expected, and *5.* results

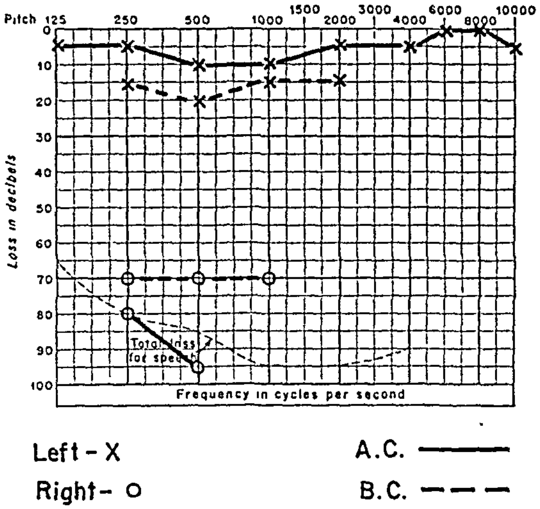


Fig. 9A. Case 11. Audiogram of patient with possible emotional deafness.

of special tests such as Doerfler-Stewart test or galvanic skin response.

Blegvad[43] added other symptoms suggestive of psychogenic deafness such as agitation and anxiety during interviews and variation in the tests on different days. Blegvad also stated that these patients usually have flat curves in the audiograms.

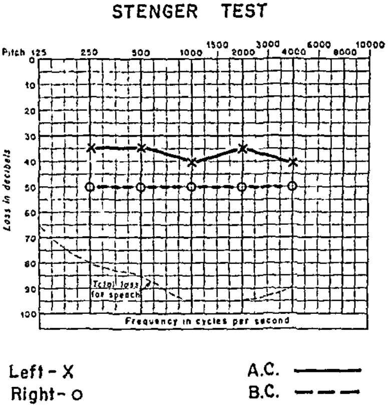


Fig. 9B Case. 11. The Stenger test Top curve represents threshold in left ear with each tone given simultaneously at 50 decibels in right. This appears to be a definitely positive finding on Stenger test.

Only one patient in my series had deafness that was due to either hysteria or malingering.

Case 11: A young married woman, aged 23 years, was in an auto accident. The next day she suddenly lost hearing in the right ear. She had no history of aural hemorrhage or dizziness. She was examined two months later. The ear, nose and throat examination gave essentially negative results. The audiogram revealed practically total loss of hearing for pure tones on the right (see Fig. 9A). The result of the Stenger test (see Fig 9B), however, was considered positive.

The patient had an insurance claim, and the case had not been settled as yet We do not know what ultimately happened to the patient's hearing.

One can only conclude from this study that, in clinical practice, psychogenic or emotional deafness of sudden onset must be rare indeed.

DIFFERENTIAL DIAGNOSIS IN GENERAL.

As mentioned earlier in this paper, sudden deafness of more or less obvious cause is not included; however, the possibility of the deafness[2] being caused by a catarrhal otitis media should be kept in mind. Often in these patients the tympanic membrane may look entirely normal or may be thickened so that no fluid is seen. The deafness, however, is always of the conduction or combined type. Often the patients give a history of having an acute upper respiratory infection at the onset of the deafness. It was surprising and disconcerting to see how many of the patients with sudden deafness of the obscure type in my series had had prolonged treatment with inflations of the Eustachian tube.

Whether sudden deafness may be caused by hydrops or vascular accident or possibly both may be impossible to determine, especially if deafness is of recent onset. One of the outstanding diagnostic points is found in the type of onset and whether the deafness is bilateral or unilateral. Bilateral onset is more indicative of Ménière's disease; the unilateral, of vascular accident. Only with careful examinations which include consultations with medical and neurologic specialists can one hope to arrive at a specific diagnosis.

GENERAL COMMENT.

Sudden deafness may be the result of any one of several conditions affecting the inner ear for which the etiology is not clearly proved. For this reason it has been a favorite area for speculation and development of theories which have been used as a basis for therapeutic procedures.

Patients who are seen immediately after the onset of deafness should be treated vigorously for Ménière's disease in the hope that irreversible changes have not already taken place in the cochlea. If hydrops is suspected to be the cause, repeated "booster treatments" seem to be of benefit.

Treatment of patients who have been deaf for two months or more and who have had no fluctuation in hearing seems useless. The results of treatment in the present series as far as hearing is concerned have been disappointing, and at least one of the reasons for this might be that the patients presented themselves late for treatment. In such a fine mechanism as that of the end-organ of hearing, irreversible changes may take place quickly. Rasmussen[6] considered the prognosis poor for the acoustic function, but favorable for the vestibular function. Hallberg and Horton[34] reported four cases of deafness in 1947 in which treatment with histamine was employed with striking improvement of hearing in one. They called the deafness "sudden nerve deafness," which is obviously a misnomer; it should have been named "sudden deafness of end-organ type."

Audiograms, loudness-balance and speech-reception tests should always be made on a patient who gives a history of sudden deafness. Of the diagnostic tests, those for hearing give the most differential information. Of greatest value are the test for diplacusis, the loudness-balance test for recruitment, and the discrimination test for phonetically balanced words.

SUMMARY.

A study was made of 178 cases of sudden deafness of obscure cause encountered in a five-year period, 1949 to 1953, inclusive. In 89 cases the deafness seemed to be due to a vascular accident. In 56 it seemed to be due to Ménière's disease. In 17 the cause could not be determined. In 15 it was due to miscellaneous causes usually of infectious origin. In one case the deafness was thought to be due to hysteria or malingering. The literature, pathology, etiology, diagnostic factors and other pertinent data were considered in some detail. Because of the absence of specific information as to the underlying pathology, the diagnosis as to etiology has to be somewhat tentative; however, in most of the cases studied, repeated and extensive examinations permitted a statement as to etiology which is considered fairly authoritative.

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NEW LOS ANGELES BRANCH FOR V. MUELLER & COMPANY.

A new retail branch store will be opened by V. Mueller & Company, Chicago, at 747 S. LaBrea, Los Angeles, Calif., the first of November. Designed to augment and improve services to West Coast surgeons and hospitals, the new branch will maintain a most comprehensive line of standard and special surgical instruments, in addition to supplies and equipment, plus direct teletype connection with the Chicago warehouse. The new operation under the direction of Mr. Hal Markham, long-time representative of V. Mueller & Company in the Los Angeles area, becomes the fifth Mueller branch strategically located about the West and Midwest. Others are in Chicago, Dallas, Houston and Rochester, Minnesota.

RECONSTRUCTION OF THE HYPOPHARYNX AND CERVICAL ESOPHAGUS AFTER RADICAL EXCISIONAL SURGERY.*

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Syracuse, N. Y.

INTRODUCTION.

Otolaryngologists are performing more radical head and neck operations each year. What is radical head and neck surgery? Generally speaking, it is the treatment of malignancies in this area with the knowledge and skill necessary to control effectively a primary lesion, a cervical metastasis, or both. *In addition,* it is the knowledge and skill necessary to restore effectively that which has been removed, so that the final result is cosmetically and functionally satisfactory for the patient. It is a combination of otolaryngeal, maxillofacial, and plastic surgery.

Recently, emphasis has been placed on excisional surgery as the primary treatment of head and neck cancers.[1,2,3,4,5] This has been greatly helped by improved anesthesia, unlimited blood replacement, and the use of antibiotics and chemotherapy. As radical surgical procedures increase, so do major problems of reconstruction. It is on these growing problems of reconstruction that increased attention should be placed, for this particular phase of head and neck surgery could be improved to rehabilitate the patient more completely. This is true in smaller communities especially, where resistance to radical surgery is based upon knowledge of a case in which reconstruction was not attempted or in which reconstruction failed.

This paper will review reconstruction techniques following radical surgery of the hypopharynx, larynx, and cervical

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esophagus. Two methods which have not been described previously and have been used successfully by us will be discussed, as well as other related problems in reconstruction.

REVIEW OF HISTORY.

The pioneer surgery for the removal of cancer of the hypopharynx and the cervical esophagus began in the Nineteenth Century when Mikulicz[6] and Czerny[7] successfully excised a carcinoma from this region. Mikulicz actually made an attempt to reconstruct the defect left from his excisional surgery. This patient died 16 months later from recurrent carcinoma. Mortality on operating was 70 per cent at the turn of the Century. Seventy-five per cent of these cases surviving surgery died of recurrent disease.[8]

In 1908 Von Hacker reviewed the literature (25 cases) and reported that his mortality rate was 48 per cent;[9] he also introduced a two stage method of closure whereby a skin lined esophagus was fashioned from two lateral rectangular flaps.

Lesions in the hypopharynx and the cervical esophagus, as stated by Lane in 1911, were one of the more challenging and difficult problems in surgery; for often a patient appeared to suffer more discomfort after surgery than before.[10] Lane attempted to repair his excisional defects by utilizing a horizontal skin flap with the bases in the left neck.

In 1913 surgeons were performing "neck gland resections" to remove large epilaryngeal lesions.[11-12] It took courage in those days to do this surgery, even though they were actually suprahyoid node dissections. If the lesion were on the anterior portion of the hypopharynx or cervical esophagus, the vertical limb of the initial incision was made as far posteriorly or laterally on the neck as possible and still obtain an exposure to the lateral pharynx; then the anterior flaps were used to cover the defect on the anterior wall of the esophagus. Conversely, if the lesion were located on the posterior pharyngeal wall the initial vertical limb of the incision was made as near the midline as possible so the skin flap could be used to cover the posterior defect in the first reconstructive stage of the lateral transthyroid pharyngotomy.[13,14] It is interesting to note that during this era emphasis was placed more on the reconstruction phase than on the excisional. For this reason initial excisional surgery often was inadequate due to the surgeon's desire to preserve tissue for reconstruction.

In 1925 Eggers reported his first case where surgical excision was used successfully.[15] No attempt, however, was made at reconstruction. It was during this period that surgeons became radical enough to expect a three year cancer cure; but still a good method for reconstructing the esophagus was lacking. Reconstruction often was prevented by complications, such as infection, shock, or lack of knowledge.

In 1926 Mackenty, a definite pioneer in surgery of the larynx, made the following statement: "I have put behind me an ambition of securing primary union." This was an expression of the times.[16]

Watson reported his first cervical esophageal case with reconstruction in 1940.[17] This patient was reported alive and well eleven years later. The esophagus was mobilized, and the lesion was exteriorized; a skin flap was then placed posteriorly to the esophagus and, during a second operation, the carcinoma was excised. A new cervical esophagus was fashioned later by further plastic surgical procedures. Watson admitted, in the critique of his method, that although successful in this instance, this method did fail in several subsequent cases. This type of surgery was not radical enough to insure against metastases to the cervical and upper mediastinal lymph nodes, and in the later cases there were recurrences at the upper or lower margins of the esophageal section and invasion of the disease into the larynx.

CURRENT TRENDS.

From 1940 to 1950 Watson, Wookey, Orton, Martin and colleagues contributed heavily to the extension of excisional surgery so that more cases were alive to be considered for reconstruction.[5,16,17,18,19] The advancement of this type of surgery was due to the courage of these men and their ability to reduce postoperative complications.

There is more agreement among the surgeons doing head and neck work today than ever before. Many believe that a minimum of surgery can be offered sometimes if the diagnosis is early; however, in most cases, where the diagnosis is late and the disease is advanced a maximum of surgery is absolutely necessary.

The patient often is left with a large pharyngeal skin fistula with saliva flowing freely onto the skin of the neck. Until this defect is repaired he is unable to swallow, must feed with a nasal gastric tube, and speech is unintelligible. If a laryngectomy is combined with excisional surgery the patient cannot attempt an esophageal voice. During this period many patients become critically depressed. Although his malignancy often is controlled or "cured," it means little to him while his swallowing mechanism is out of order.

TABLE I.

SUMMARY OF RADICAL EXCISIONAL OPERATIONS MENTIONED IN LITERATURE.*

1877-1955.

		Cases**
A.	Partial pharyngectomy and/or cervical esophagectomy conserving the larynx.	1
B.	Partial pharyngectomy and/or cervical esophagectomy with total laryngectomy.	1
C.	Partial pharyngectomy and/or cervical esophagectomy, total laryngectomy with thyroid lobectomy combined with radical neck dissection.	4
D.	Partial pharyngectomy and/or cervical esophagectomy, total laryngectomy, total thyroidectomy combined with bilateral neck dissection.	1
E.	Total pharyngectomy and/or total cervical esophagectomy, total laryngectomy and total thyroidectomy.	4
F.	Total pharyngectomy and/or total cervical esophagectomy, total laryngectomy, total thyroidectomy combined with radical neck dissection.	4
G.	Total pharyngectomy and/or total cervical esophagectomy, total laryngectomy, total thyroidectomy combined with bilateral neck dissection (either one or two stages).	9
	TOTAL	24

* References: 1-13.

** Our group.

Many of these defects are repaired by a simple layer closure. When bilateral neck dissections have been performed, and there is a good deal of postoperative scarring of the neck, and in some cases with scarring effects of heavy radiation therapy, the problems appear extreme. It is in these cases that the ability, training and experience of the surgeon make the difference. It is here where the individual problem presented often is solved by the ingenuity of the operating surgeon.

Table I represents various procedures described in the literature which are between the maximum (for advanced cases) and the minimum (for the very early cases). Table II represents the methods of reconstruction which have been described in the current literature together with two new methods which I used successfully in two instances. Each will be described in detail. The advantages and disadvantages of each will be discussed together with my observations in using these procedures.

TABLE II.

SUMMARY OF METHODS OF RECONSTRUCTION MENTIONED IN LITERATURE.*

1908-1955.

		Cases**
A.	Anastomosis with neck flexed or transverse closure	7
B.	Skin tube and double sliding flaps.	3
C.	Migration of tube pedicles.	5
D.	Wire mesh-skin graft.	2
E.	Migration of flat pedicle.	1
F.	Use of tongue as pedicle.	1
G.	Died before reconstruction.	5
	TOTAL	24

* References: 9, 13-15, 17-19, 22-30.
** Our group.

SURGICAL METHODS.

End-to-End Anastomosis is the least complicated method, technically, and the one most frequently used to effect reconstruction. It usually is successful if used when indicated, as in cases where the primary lesion is in the hypopharynx or extrinsic larynx and has spread by direct extension to the uppermost part of the cervical esophagus. The spread to the cervical esophagus in these instances is usually an early, well defined, direct extension of the original lesion and appears resectable by including a segment of the cervical esophagus. A radical neck dissection may or may not be combined with this procedure depending upon the circumstances.

The repair used in these cases is the same as used by general surgeons in the repair of intestinal defects when a segment of small or large bowel has been resected. Its advantage lies in the principle of the transverse closure which prevents stenosis and complicating strictures after the healing is complete. For many years laryngologists closed these hypopharyngeal defects in a vertical line. This lessened the potential diameter of the repaired esophagus, since the greatest circumference at the point of reconstruction could never exceed the width of the mucous membrane left on the posterior esophageal wall. This was recognized by Jackson, Clerf,[20] and Cunning[21] who utilized a "T" closure. The "T" closure increased the diameter at the point of reconstruction and later evolved into a horizontal or transverse closure which gives the greatest diameter. Laryngologists today are very successful in effecting a closure even when as little as ½ cm. of mucosal strip remains. Usually these heal without the complication of fistula formation.

The utilization of the transverse closure enables surgeons to increase the potential diameter at the time of closure so it is rarely ever smaller than the original esophageal diameter. It is closed in layers with a 3-0 or 4-0 atraumatic chromic intestinal suture. A continuous suture is placed inverting each stitch so that the mucosa is always turned towards the lumen and approximates itself. This makes a tight seal. The first suture line then is reinforced by a second layer of interrupted sutures covering the entire length of this first suture layer.

If it is not possible to save a mucosal strip[22] then the direct end to end anastomosis is necessary. This is possible in selected cases where the neck is thin and the surgeon is able to mobilize the distal segment and pull it superiorly so that a direct transverse anastomosis of the distal proximal ends of the esophagus is obtained. A flexing of the neck greatly aids in closure. With the help of the orthopedic department we have made a light head case to hold marked flexion for seven days and found that primary healing was greatly facilitated. If this anastomosis fails, as it could if the resected segment of the cervical esophagus is more than 2 cm., it still will have held for a minimum of five to six days. By this time the radical neck flaps are well sealed and reconstruction is far less difficult than if the alternate method of closing with pharyngostoma and esophagostoma were used. A disadvantage of this alternate method is that salivary secretions will flow over the fresh neck wound and primary healing of the skin flaps often is delayed. Some cases will close spontaneously if the complicating salivary fistula is not too large, and the distal drainage is not impeded by stenosis at the point of anastomosis.

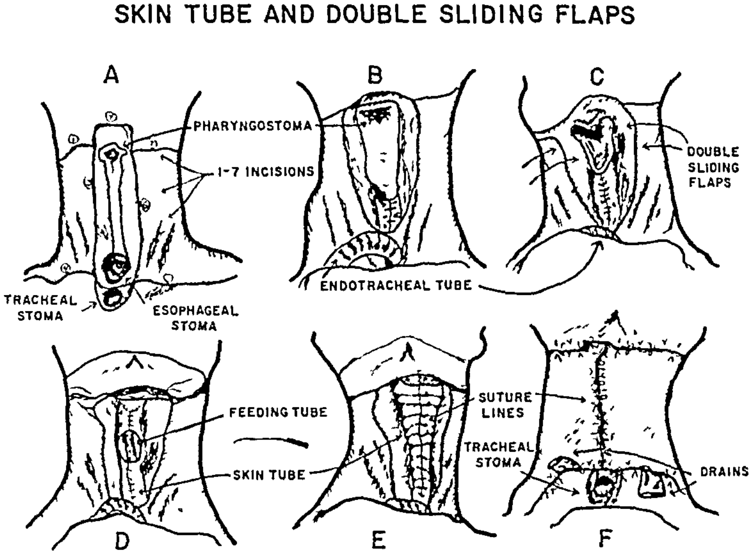


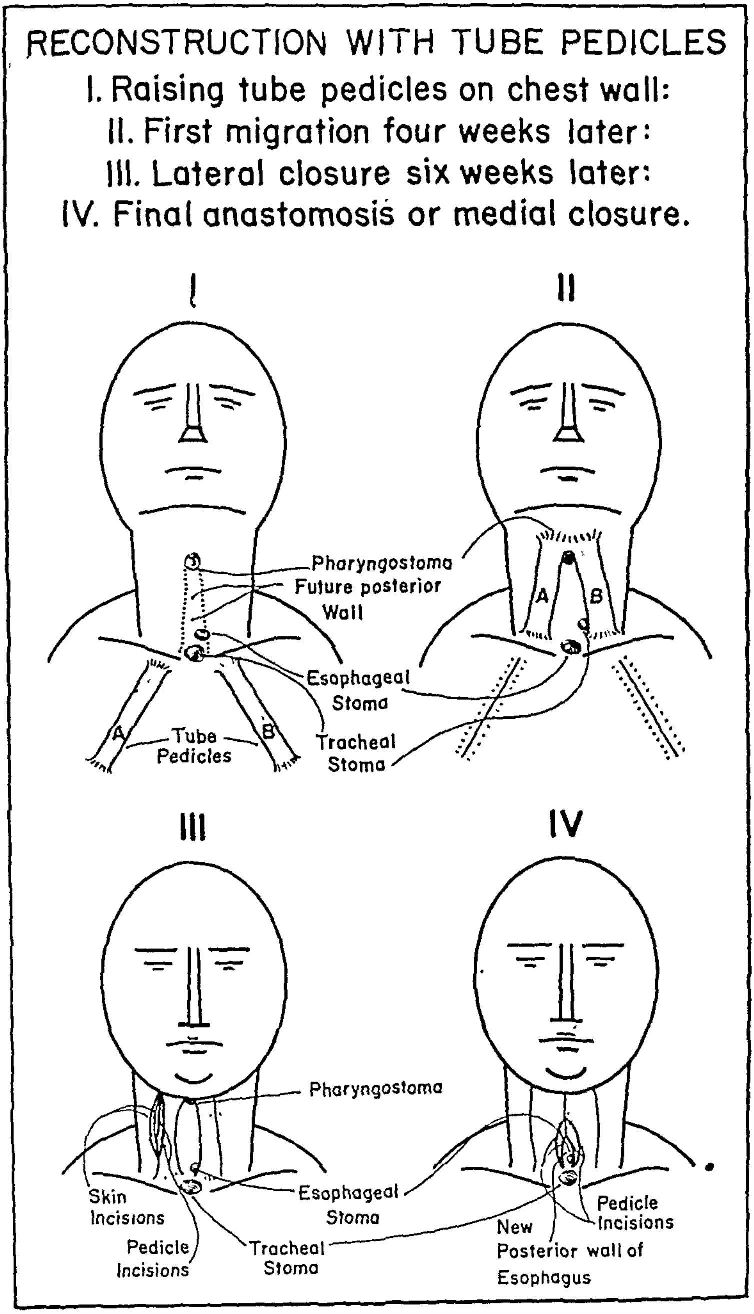
Fig 2

The Skin Tube With Double Sliding Flaps, as described by Watson,[23] is today one of the most used reconstruction procedures of head and neck surgeons. Its application is broad, and the final result usually is excellent. It is used when excisional surgery leaves a pharyngostoma and an esophagostoma. The skin between the pharyngostoma and the esophagostoma is healthy enough to withstand mobilization and multiple incisions. (See incisions 1 to 7 as diagrammed in Fig. 2A. The central island of tissue left connecting the pharyngostoma with the esophagostoma is rolled onto itself (see Fig. 2B). Then the two lateral rectangular flaps are well undermined (see Fig. 2C) ; a feeding tube is put in place, and the first row of sutures is completed (see Fig. 2D). Fig. 2E shows the reinforcing line of sutures over the initial suture line. The two lateral rectangular flaps are mobilized in such a way that it is possible to bring them to the midline and close them under tension. Drains are placed (see Fig. 2F). It is important that one of the rectangular flaps be made slightly longer than the other in the final reconstruction so that the outside suture line is located to one side of the inner suture line and not in exact contact. This lessens the possibility of fistula formation.

The advantages of this method of reconstruction are obvious. In a one-stage technique it usually is possible to undertake and complete reconstruction three to four months after the original excisional surgery. Often it could be done before this time, but it seems wise to wait to be certain that residual tumor is not present. The waiting period is advantageous, too, since the skin quality improves during this interval and increases the likelihood of successful reconstruction.

This method can not be used when secondary healing from a postoperative wound infection causes marked scarring in the neck. The tissues in the neck then may be of such a quality as not to withstand the amount of plastic work necessary in this one one-stage procedure. This particularly is true if the patient has undergone a laryngectomy followed by a bilateral radical neck done in two stages. This type of surgery leaves the neck with little reserve tissue. Heavy radiation prior to excisional surgery is also a contraindication to the use of this method. When the surgeon is faced with either of these situations it is best to consider the migration of healthy tissue to the neck from a distant donor site such as the chest or abdomen. This method we will discuss next.

The Use of a Single or a Bilateral Double Tube Pedicle Graft[24] to bring good tissue from an hairless portion of the anterior chest to the neck has played an important part in the reconstruction of many of these cervical, esophageal, and hypopharyngeal defects during the past several years (see Figs. II, III. Many of these defects could not be closed before



RECONSTRUCTION WITH TUBE PEDICLES

I. Raising tube pedicles on chest wall:
II. First migration four weeks later:
III. Lateral closure six weeks later:
IV. Final anastomosis or medial closure.

Fig. 3

this method was employed, and these patients became dependent upon feeding tubes for the rest of their lives.

As indicated above, most of these patients had maximum neck surgery which usually means that they have had a larynx, cervical esophagus, thyroid gland and parathyroid glands and contents of both lateral necks removed (see Fig. 4). Often it was necessary to remove a large segment of skin in order to encompass the tumor. In addition, some patients had had heavy radiation to the neck at some time or another, and in these latter cases metabolism usually is controlled by the use of replacement therapy; that is, large doses of vitamin D, calcium, and thyroid extract. All these factors present complications in plastic surgery.

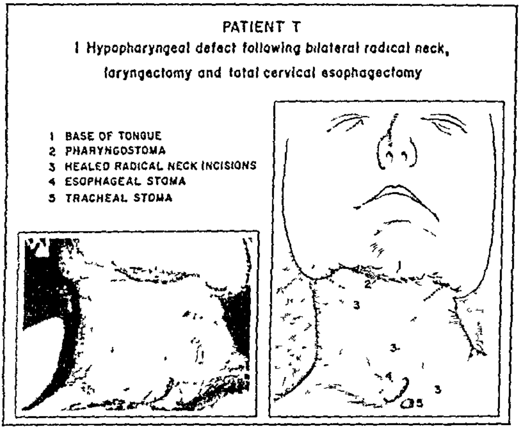

PATIENT T
I Hypopharyngeal defect following bilateral radical neck, laryngectomy and total cervical esophagectomy
1 BASE OF TONGUE
2 PHARYNGOSTOMA
3 HEALED RADICAL NECK INCISIONS
4 ESOPHAGEAL STOMA
5 TRACHEAL STOMA
1
2
3
3
3
4
5

Fig 4

Often the pharyngostoma itself complicates the wound healing of the tube pedicles, because it is a likely source of infection (see Fig. 4). Great attention to wound care by the nursing staff, the resident, and by the attending staff is necessary during the period when tubes are being migrated from the chest to the neck. In several cases we intentionally closed the pharyngostoma prior to migrating the tube pedicles to remove this problem of contamination when the pedicles were transferred. Once the tubes were migrated and an adequate blood supply was established, the pharyngostoma was opened. Frequently these patients are elderly, and since they are in an hypothyroid state, primary wound healing is very slow.

It is the clinical experience of our group that tissue resistance in these tube pedicles is low and directly related perhaps to the loss of the thyroid and parathyroid glands. We are sufficiently concerned to have started research in rats to clarify this situation. Few conclusions can be drawn to date, although we have done thyroidectomies on 50 rats and have compared their primary wound healing with that of normal rats.

We feel certain that even though adequate replacements for thyroid and parathyroid hormones are instituted, a certain percentage of wounds demonstrates a lower resistance to infection and primary healing. The two pedicle grafts often appear healthy for as long as seven days and then take on a localized gangrene about the suture lines. We are most interested in studying this phase of wound healing in these particular problems. It has been our experience that before a migration is made, and before the actual transplantation is undertaken, the surgeon must be certain of the circulation in the flap. Any simple method whereby the circulation in the pedicle flaps can be occluded gradually might be helpful and could save the patient and surgeon the chagrin of losing all or part of the flap after several operations have been performed.[25] Necrosis of any sizable amount can be most depressing to these patients and creates a difficult situation. If we encounter circulatory distress in the flap it is our policy to delay the separation by at least a week. For this reason, we have no standardized time for migrating the flaps. We try to make migrations approximately three weeks apart once we start this type of reconstruction; but we are frequently delayed because of circulatory distress. We have been successful in using this double tube method in several different types of cases.

We fashion bilateral or single, double tube pedicles on the anterior chest wall and at three-week intervals migrate them to the defect in the neck for reconstruction. Sometimes it is possible to use just a single double-tube pedicle, particularly in cases where just one side of the neck is badly scarred. Normally the use of the two pedicles is more satisfactory, for we can effect reconstruction so that the patient can swallow and, in time, develop an intelligible esophageal voice.

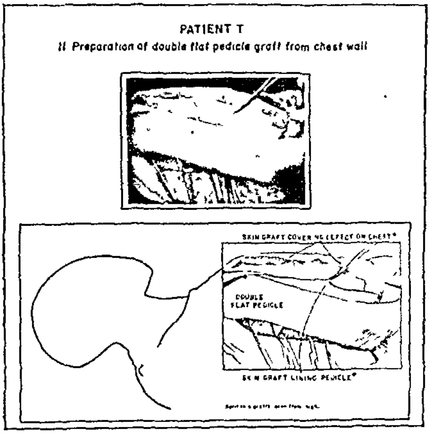
The Wire Mesh and Skin Graft is a technique used frequently by some surgeons.[12,26,27,28] Primary reconstruction of the esophagus at the time of excisional surgery has been effected by bridging the gap between the proximal and distal ends of the cut esophagus with a wire mesh tantalum or stainless steel mesh which is prepared at the time to fit the defect. A polyethylene tube has been used by the British.[29,30] The wire mesh is rolled to form a cone resembling the hypopharynx and cervical esophagus. Then the mesh is covered by a thin split thickness skin graft taken from the thigh or abdomen with a Reese or Padgett's dermatome. The skin is sutured to this wire mesh in such a fashion that the epidermal side of the skin graft faces the lumen of the esophagus; the wire mesh acts as a support for the skin graft. The ends of the tantalum cone covered by the graft are sutured to the proximal and distal ends of the esophagus and the cone is placed into the neck. The vertical cut ends of the skin graft should be in immediate contact with the prevertebral fascia; this helps in preventing the formation of fistulae. The method was described originally (1949) by Rob and Bateman and is referred to in literature as the one-stage tantalum mesh method for reconstruction of an excised cervical esophagus.[31] These grafts heal very nicely, and there are numerous reports in the literature of rapid and complete functional rehabilitation of the esophagus. The tantalum or stainless steel mesh is removed as soon as the epithelization is completed; usually this is within four to six weeks. It is possible to view the area by the use of a hypopharyngeal mirror; a direct esophagoscopy may be performed if the surgeon is not certain that anastomosis is complete. The mesh is easy to remove by using strong alligator forceps and gently teasing it away from the new esophageal lumen. It is then crushed and removed endoscopically. At this stage the patient is swallowing. Swallowing begins in ten to twelve days, usually depending upon the appearance of the external healing of the neck. Closure over the mesh is similar to any closure, except one flap should be longer so that the suture line is lateral to the center of the wire mesh skin graft cone.

We used this method in two cases but did not find it as satisfactory as other surgeons reported. In one case the mesh appeared to act as a rigid foreign body causing necrosis by sheer pressure. This necrosis led to a severe localized infection and cellulitis; it was necessary to transfer a chest flap to the area to effect a closure. Edgerton reported this same objection in 1952 and Conley in 1953.[27,28]

In a second case, residual or recurrent disease became apparent within six months after the graft had healed and the patient had established function. It was our feeling that, in this particular case, perhaps the residual or recurrent disease near the prevertebral fascia had been masked or buried by this rather bulky reconstruction which overlay the site of the disease. This type of recurrence with the wire mesh method was reported by Rob and Bateman in their original three cases;[31] furthermore, we have seen cases where residual disease became evident after a pharyngostoma and esophagostoma were formed and before reconstruction was started. In one of these cases we were able to control the residual disease by performing a radical neck dissection on the opposite side. We felt that the residual disease was diagnosed much earlier than had we covered the area with a wire mesh cone and skin graft. In recent literature VonDielin has taken exception to this method and stated emphatically that one should not worry about recurrences since the return of function and appearance means much more to the individual patient.[32] He feels that primary reconstruction should be attempted as soon as it is at all feasible. Our group readily admits that some patients do become very depressed. Often there is a genuine problem of management when patients must ride through the multiple stages of reconstruction. For this reason we have tried to discover, with the help of the animal laboratory, why some of the multiple procedures fail. We have tried to evaluate each individual problem as it presented itself and have found two additional techniques which have been successful in speeding up the reconstruction period in the cases to which they were applied. These new methods will be discussed next.

OUR INNOVATIONS.

The greatest objection to the use of the bilateral double-tube method is that it is a five to ten stage procedure at best and when finally completed the neck is a maze of suture lines.[33] It is then possible, during healing, for these suture lines to contract and infringe upon the diameter of the reconstructed esophageal lumen. In addition, it always seemed necessary for us to raise two tube pedicles, thus doubling the operative morbidity and complications.


PATIENT T

II Preparation of double flat pedicle graft from chest wall

SKIN GRAFT COVERING DEFECT ON CHEST*
DOUBLE FLAT PEDICLE
SKIN GRAFT LINING PEDICLE*

Fig. 5

In an attempt to simplify these stages, we felt that a single broad double pedicle might be more satisfactory than a bilateral pedicle brought to the midline and then anastamosed. It seemed reasonable to use a flat pedicle rather than one tubed or circular, for it would mold to the neck and lend itself to ally depending upon the appearance of the external healing of the neck. Closure over the mesh is similar to any closure, except one flap should be longer so that the suture line is lateral to the center of the wire mesh skin graft cone.

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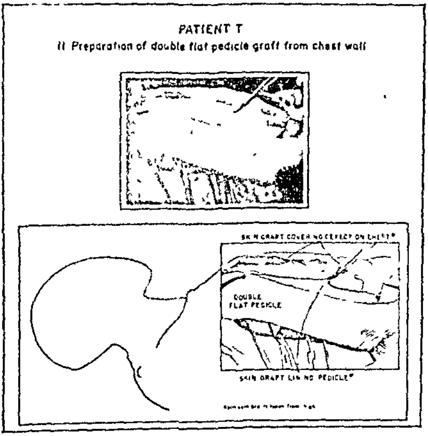
PATIENT T
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PATIENT T.

III. Third migration of pedicle now in place between pharyngostoma and esophageal stoma.

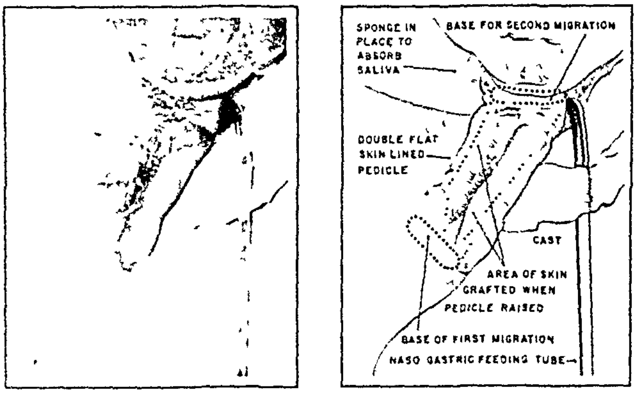
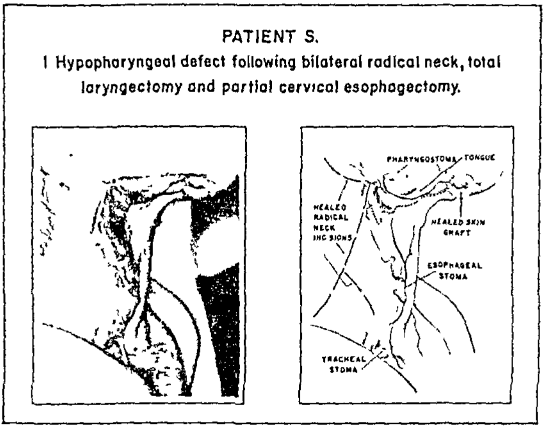

SPONGE IN PLACE TO ABSORB SALIVA
BASE FOR SECOND MIGRATION
DOUBLE FLAT SKIN LINED PEDICLE
CAST
AREA OF SKIN GRAFTED WHEN PEDICLE RAISED
BASE OF FIRST MIGRATION
NASO GASTRIC FEEDING TUBE→

Fig. 6

in the neck so that its distal end was just below the esophagostoma. The lateral ends of the flat lined pedicle were sutured to the skin in layers on either side as in the bilateral double pedicle (see Fig. 3-4). This method of reconstruction proved very successful.

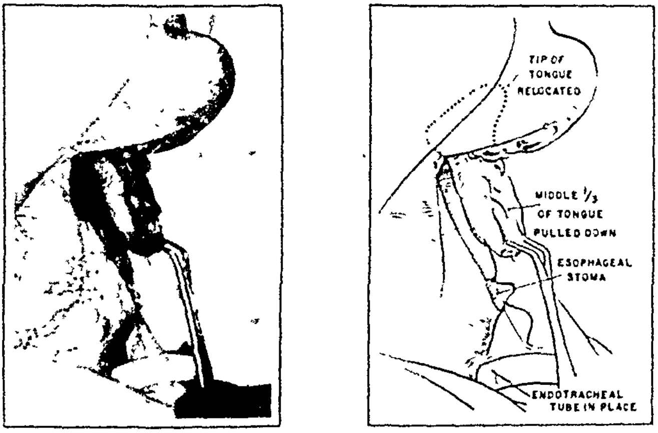
In another instance, where the excisional surgery included not only the larynx and cervical esophagus but also the posterior third of the tongue, the defect was more hypopharyngeal, and the center neck was markedly scarred. In this case a different approach was used. The original lesion in the hypopharynx was most extensive. In removing the posterior one-third of the tongue both hypoglossal nerves were severed. The remaining part of the tongue was used to fill the defect in the submental space forming a large pharyngostoma (see Fig. 8). A split thickness graft was used to help close the skin to the mucous membrane where it had been necessary to excise widely to remove skin involved with recurrent disease (see Fig. 8). Our biggest problem was to find tissue to form


PATIENT S.

1 Hypopharyngeal defect following bilateral radical neck, total laryngectomy and partial cervical esophagectomy.

Fig 7

the anterior wall of the hypopharyngeal defect. In this particular case we could have raised a large double-tube pedicle. The skin on the lateral neck was soft and pliable and could be used for the posterior wall. At this point we conceived the idea of using the base of the tongue (in reality the middle third, since a posterior one-third had already been removed). The tongue mobilized very well; it was already functionless and no problem to the patient. It was possible, by pulling, to bring the middle third of the tongue down as low as the esophagostoma (see Figs. 7 and 8). This was done by reducing the thickness of the mucosa and mobilizing both sides of the floor of the mouth (see Figs. 7 and 8). The blood supply was excellent at all times. The anterior wall of the reconstructed defect was formed by using a modified "Z" plasty, and the scarred bilateral neck flaps were transferred to cover the entire defect (see Fig. 9). Although a portion of these external flaps sloughed out postoperatively, it was evident from the beginning that the inside layers were going to heal. This, I believe,

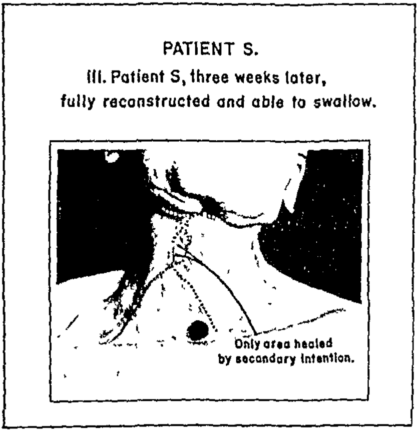


PATIENT S.

II. Using tongue to form reconstructed anterior wall of hypopharynx and cervical esophagus.

Fig. 8

was due to the marked vascularity of the tongue tissue used to form the new anterior wall of the esophagus. It was a matter of seven to ten days before epithelization of the sloughed area external to the tongue began. The patient was able to swallow about three weeks after this one-stage reconstruction. By utilizing the tongue tissue I feel we saved the patient four to five months of intermittent hospitalization and several operative procedures.



PATIENT S.

III. Patient S, three weeks later, fully reconstructed and able to swallow.

Only area healed by secondary intention.

Fig. 9

EXPERIMENTAL STUDIES.

Animal surgery already has contributed somewhat towards the advancement of esophageal and tracheal reconstruction work.[34,35] At present we are trying to grow tracheal homographs of dogs in their abdomens. We are trying to grow them in their peritoneal cavities and are trying to cover the inner surface of the tracheal homographs with intestinal mucosa. Our thought is that if these homographs grow we will transplant them to the neck by multiple stages in order to build a better esophagus. We have experimented considerably with tantalum and wire mesh together with polyethylene tubes and skin grafts. We feel that the use of soft sponge rubber as a splint is far superior to the rigid splints. It has been mentioned that rigid splints may cause pressure necrosis. We believe we have solved one of the more difficult problems found in reconstruction work in dogs. We have constructed a tracheotomy tube patterned after a tracheotomy tube which is inserted into horses when they are racing.

As far as we know this is the first time this type of tube has ever been used in dogs. We were able to keep a dog alive for two months with this tube. Previously, most of our dogs died from two to three weeks with the conventional type tube or when the trachea was exteriorized to the skin. Other investigators have successfully used a polyethylene tube as a tracheal implant. In four months a fibrous lining grew into the tube.[36] Our enthusiasm for animal reconstruction work in this area becomes more vigorous each year as progress is noted. We are hoping that it will be possible to reconstruct a larynx. Certainly the reconstruction of half a larynx has been successful.[37]

SUMMARY.

In reviewing the literature it becomes apparent that radical surgery for the control of advanced hypopharyngeal, laryngeal, and cervical esophageal malignancies is not a new method of treatment. Large defects in the continuity of the pharynx and the esophagus have been a necessary result of this type of surgery since the first operations in the Nineteenth Century. Little attention was given to reconstruction problems by early surgeons and their morbidity and mortality rate was prohibitive. Excisional surgery was inadequate, and recurrent or residual disease eventually took its toll from the few apparent successes.

Between 1920 and 1926 more attention was given to the reconstruction and rehabilitation phase of head and neck surgery. The number of complete cures remained low, because surgery was still inadequate. In recent times excisional surgery has become a primary method of treatment for advanced malignancies in this region. Larger and more complicated defects a[illegible]lting, and reconstruction is becoming increasingly co[illegible]ted. Since reconstruction of these defects is essential and highly individualized, every successful technique should be [illegible]orted.

CONCLUSIONS.

1. Four reconstruction methods used successfully by others have been tried by our group. These procedures are described and compared. *Our experiences and observations with each* method are discussed.

2. Two additional methods have been used recently by our group with apparent success. These procedures are described and discussed.

3. Experimental work in animals (dogs and rats especially) has been started. This work can be exceedingly helpful in studying the problems of reconstruction and in applying any new techniques.

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A SPECIAL POSTGRADUATE COURSE.

An introduction to fundamentals of reconstructive surgery of the nasal septum and external pyramid will be presented by Yale University School of Medicine Department of Surgery, Section of Otolaryngology, in cooperation with The American Rhinologic Society and Grace-New Haven Community Hospital, May 11 - 18, 1957.

The course will be given under the guest direction of Dr. Maurice H. Cottle. It will consist of didactic lectures, case presentations, cadaver dissections, and observation of operations. All sessions will be held at the Yale-New Haven Medical Center, New Haven, Connecticut, U. S. A.

Enrollment will be limited. Applicants must be diplomates of the American Board of Otolaryngology, or its equivalent for doctors from other countries. Scholarship assistance is available to a limited number of foreign applicants.

For further information write to Assistant Dean of Postgraduate Medical Education, Yale University School of Medicine, 333 Cedar Street, New Haven 11, Connecticut, U.S.A.

IDIOPATHIC HEMOTYMPANUM.

REVIEW OF THE LITERATURE AND REPORT OF THREE CASES.*

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Judging from the paucity of reports in medical literature, idiopathic hemotympanum is uncommon. The condition is mentioned in only one[1] of the current textbooks that we have reviewed. Since 1951, three cases have been treated in this hospital: the first by simple mastoidectomy; the second by radical mastoidectomy; and the third by simple mastoidectomy, followed by two attempts to produce a permanent perforation in the tympanic membrane.

In 1952, Menck-Thygesen[2] reviewed the literature and listed 11 cases, including two of his own. The diagnosis on two of these[3] was not definitely established, myringotomy was not performed, and the character of the fluid in the middle ear of either of these two cases was not known. Since the condition completely cleared up following adenotonsillectomy, serous otitis media appears to be a more plausible diagnosis. In this latter condition it is not uncommon for the tympanic membrane to present a dark bluish cast, similar to the color seen in hemotympanum.[4] Baron speaks of this color as "gun-metal blue".[5] We, therefore, feel that these two cases should not be classified as Idiopathic Hemotympanum.

One case of blue eardrum, discussed in considerable detail by Johnston,[6] in his paper on idiopathic blue eardrum, was described by Vior.[7] The discoloration was limited to the posterior inferior quadrant of the drum membrane, a definite pulsation was visible, and there was a pronounced bulge which

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increased in the blue area when the homolateral jugular vein was compressed. Vior concluded that his case was one of jugular bulb protrusion. This is another case that should not be listed under the title, Idiopathic Hemotympanum.

Johnston,[6] in 1953, reported two cases. After eliminating the three cases previously mentioned (O'Donnell and Vior) as cases that should not be classified as Idiopathic Hemotympanum, the total number of cases previously reported is 11. The three cases herein reported bring the total up to 14 at the time of this writing.

CLINICAL PICTURE.

In the cases reported both male and female have been affected, and the ages range from eight to 60. In no case have both ears been affected. The patients have come for treatment because of pain, discharge or hearing loss. If pain is present, it is usually intermittent and suggests Eustachian tube closure.

The pain in some cases has been associated with acute otitis media. The hearing loss is conductive, the only unusual aspect being that in some cases it has been much greater than is usually seen with fluid in the middle ear. If discharge has occurred, it may be purulent (from acute suppuration), or reddish brown (chocolate colored). One case[2] developed immediately following acute suppurative otitis media.

Examination of the patient may reveal nothing of significance other than the otologic findings on the affected side. The tympanic membrane is intact (if discharge has not occurred recently) and might suggest serous otitis media on first glance, because of the bluish color; but it can be seen that it is not the gun-metal blue of serous otitis, but rather a definite bluish-black suggesting hemotympanum. In a number of the cases the tympanic membrane has appeared to be retracted despite a patent Eustachian tube, and did not move perceptively with inflation. The Eustachian tube has been open in the majority of cases, resulting in the conclusion that Eustachian tube closure probably is not a factor.[8] The most important finding from a standpoint of diagnosis is the character of the fluid found in the middle ear. All reported cases subjected to myringotomy have shown a fluid that appeared to have come from a liquified hematoma. This is reddish-brown, chocolate-like, and often moderately viscous. Detailed laboratory examinations[2,8] have shown this fluid to be sterile blood, or blood products. Roentgenograms of the mastoid usually show a pneumatized bone with clouding of the air cells, fuzziness of the intercellular septa, and may show bone destruction. Some cases have had a sclerotic mastoid.

DIFFERENTIAL DIAGNOSIS.

Johnston[6] goes into detail on the various conditions which may cause a "blue eardrum". These include hemotympanum due to head injury, barotrauma, or hemorrhagic diathesis; vascular anomalies, such as varices of the middle ear or protrusion of the jugular bulb; and conditions resulting from obstruction of the Eustachian tube such as the rupture of a hemorrhagic bleb in acute otitis media, or serous otitis. Serous otitis media is seen more often than any of the other conditions mentioned above, but if the tympanic membrane is blue, it is a gray-blue or gun-metal blue, and not bluish-black. Because of the confusion that is fostered by the use of the term "blue eardrum"[3,6] as synonymous with idiopathic hemotympanum, we suggest that the former term be dropped. We agree with Johnston, who mentioned that the most important diagnostic finding was the chocolate-like fluid found on myringotomy.

TREATMENT AND RESULTS.

When first seen most cases have been treated similarly to serous otitis media, with myringotomies and Eustachian tube inflation. This has never resulted in cure of the condition as far as is known. Menck-Thygesen[2] noted in his cases that repeated paracentesis and inflation led to progressively less and less improvement in hearing, and finally no improvement. He also noted that the tympanic membrane became anesthetic in time. These findings were not present in our cases.

Of the 11 cases of idiopathic hemotympanum reported, one[9] received no treatment, and five[2,6,10,11] were treated by repeated inflations and myringotomies with only fair to disappointing results. Tubal obstruction was felt to be a factor in one case;

obstruction was not commented upon in two; and in two the Eustachian tube was patent.

The treatment and results were not stated in one[1] case.

The remaining four cases were subjected to simple mastoidectomy. Ranger[8] operated on a child and a young adult. Neither had tubal obstruction as a factor, and prior to mastoidectomy repeated myringotomies and inflations were carried out, but without improvement. Mastoid X-rays showed evidence of disease other than simple cloudiness due to fluid, and at surgery the mastoids were filled with the same chocolate-like viscous material that had been obtained by myringotomy. There was no bone destruction. Analysis of the fluid showed no bacterial growth, and that it was essentially altered blood. Microscopically there were heavy cholesterol crystal deposits, multinucleated giant cells, and "cholesteatomatous tissue" without squamous epithelium. Both patients had a normal tympanic membrane post-operatively, and the child's hearing was markedly improved—an 11 db loss in the speech range.

Menck-Thygesen[2] operated on a ten-year-old boy who developed idiopathic hemotympanum following an acute suppurative otitis media. There was no persistent tubal closure in this case after the condition developed, although it was assumed that tubal closure was, in part, responsible for the preceding acute otitis media. The "viscous brownish-red fluid" was sterile, and was shown to be a product of blood. Roentgenograms showed a well pneumatized mastoid with blurred cell walls and some bone destruction. At surgery the mastoid was filled with a fluid and a type of granulation tissue similar to that described in Ranger's cases. Post-operatively the hearing and tympanic membrane returned to normal.

In 1953 Johnston[6] reported a case of a boy, first seen at the age of eight, with an acute suppurative otitis media, one month following an adenotonsillectomy. On penicillin therapy he made an uneventful recovery and was seen periodically thereafter. The tympanic membrane was normal, and the Eustachian tube open. A 35 db conductive hearing loss, which did not change with tubal inflation, developed. One year following

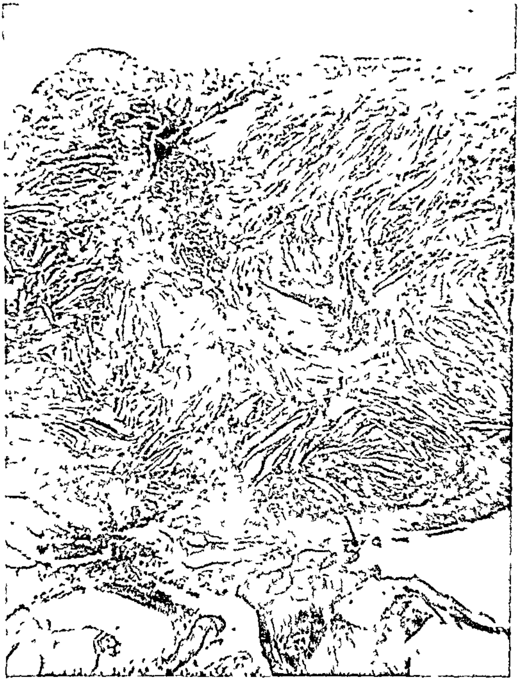

Fig 1. Low power view

the acute episode the drum membrane took on the appearance of an unresolved blood clot. During the following four years the color of the tympanic membrane and the hearing loss remained the same, and there were three or four episodes of spontaneous otorrhea of thick reddish-brown material, all of which subsided with penicillin therapy. Roentgenograms demonstrated a well-pneumatized mastoid with diffusely increased density, and a fuzzy altered trabecular pattern. Five years after the initial episode a simple mastoidectomy was per-

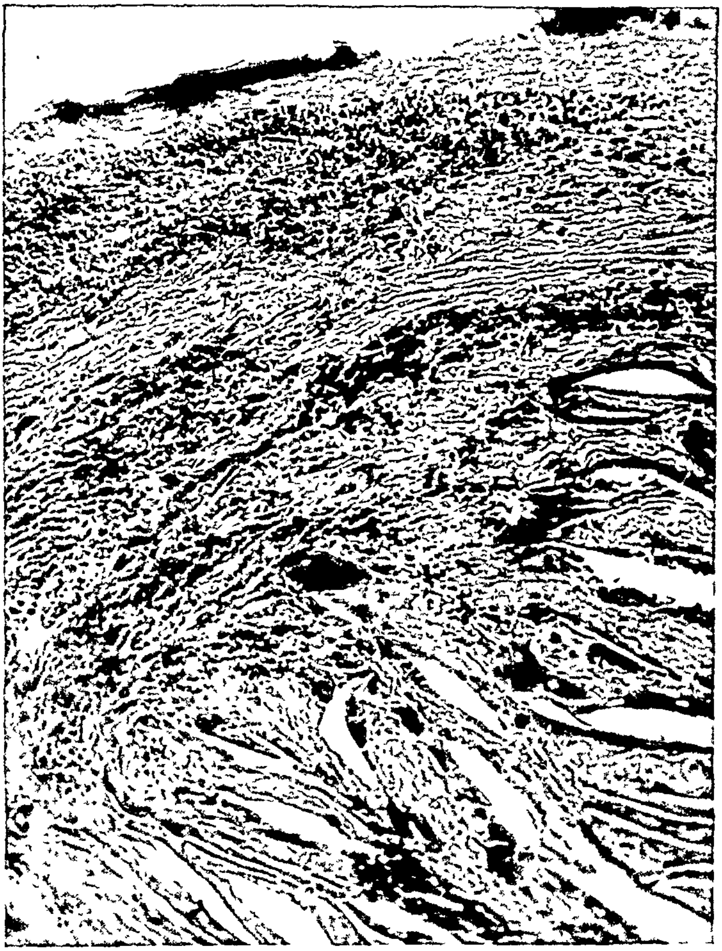

Fig. 2. High power view.

formed. All cells were filled with sterile, thick reddish-brown material, and there was a suggestion of pressure necrosis of bone in the antrum. Tissue removed at surgery showed fibroblastic proliferation, chronic granulation tissue with cholesterol crystals and foreign body giant cells. Six weeks following surgery there was a marked hearing improvement, although there was still a minute amount of fluid in the middle ear, and a reappearance of the blue-black coloring of the eardrum.

The reason for performing simple mastoidectomy in these cases are three: first, it appears that there is some disease process going on in the mastoid cells themselves, and mastoidectomy is a logical approach to a cure, even though the cause is not known. Second, whatever the disease, the mastoid space seems to be acting as a reservoir for fluid, and its obliteration is logical as an approach to cure. Finally, there is the possibility that there is some minute bleeding area somewhere in the mastoid, and, although it may not be seen, an obliteration of the mastoid cavity might bring a halt to the disease or manifestations of the disease.

Of the four cases subjected to simple mastoidectomy, three were cured of their disease process. The fourth case[6] had a marked hearing improvement, but persisted in forming fluid, and maintained the blue-black eardrum.

ETIOLOGY.

Nothing is known of the cause of this condition. Most writers who have discussed it have come to the conclusion that it is best called idiopathic. It seems likely that somewhere in the mastoid, tympanum, or Eustachian tube there is a small area that bleeds. Surgery has not uncovered the source thus far.

Pathologically all cases coming to mastoidectomy, including the three cases presented herein, have shown the same general nonspecific picture: fibrous connective tissue containing numerous small blood vessels, chronic inflammatory cells, cholesterol clefts, and a variable number of foreign body giant cells (see Figs. 1 and 2). Cultures of the viscous reddish-brown fluid that is found have been negative.

CASE REPORTS.

Case 1: A 17-year-old white boy was first seen at this hospital in March, 1951, because of progressive hearing loss and recurrent attacks of drainage from his right ear. An adenotonsillectomy had been performed in 1941. He first had ear trouble in 1948, and since that time had five attacks of what sounded like uncomplicated acute suppurative otitis media, all but one attack being in the right ear. In 1949 a re-adenoidectomy was done, followed by radium irradiation to the nasopharynx. At that time the record shows that both tympanic membranes were intact; no mention was made of discoloration. He continued to have a slowly progressive hearing loss and attacks of otitis media. His general health was good.

On examination the nasopharynx showed some regrowth of adenoids, especially in the fossae of Rosenmueller. There was a thin, reddish-brown discharge issuing from a central perforation of the right eardrum, the result of an acute otitis media. After two weeks the discharge had ceased (penicillin was given during part of this time), and the drum membrane is recorded as being black and retracted.

In April a re-adenoidectomy was done, but this had no effect on the general picture. There was an average loss of 33 db in the speech-hearing range.

Examination in July revealed no change in the hearing. The Eustachian tube was inflated with ease. The right eardrum was described as shiny and black, with a tit-like projection postero-inferiorly and a bleb-like projection at the pars flaccida. Roentgenograms revealed a well pneumatized mastoid with diffuse clouding. Simple mastoidectomy was performed* revealing a mastoid filled with slightly viscous reddish-brown fluid, and dark granulation tissue. The antrum was free of this granulation tissue.

Histologic examination of this tissue revealed a fibrous connective tissue in which there were numerous cholesterol clefts surrounded by multinucleated giant cells. There were scattered areas of hemorrhage and chronic inflammatory cells.

The post-operative course was uneventful, and examination of the ear 15 days later revealed a persistence of the black coloring of the tympanic membrane.

Follow-up examination performed by Capt. W. R. Green at Fitzsimmons Army Hospital, March 21, 1955, showed the tympanic membranes to be essentially normal and audiogram showed no hearing loss.

Case 2: A 20-year-old man of Japanese descent was first seen at this hospital in January, 1953, because of a two-week episode of "bloody discharge" from his left ear three months previously.

He had first noted hearing loss in his left ear seven years previously, and believed he had had some discharge from the ear three years prior to that. He had intermittent earaches for three years, and tinnitus for two years. Three months prior to hospital admission he developed pain in his ear which was relieved by spontaneous discharge of a bloody material.

Examination revealed the nasopharynx to be relatively free of adenoid tissue. The right ear was normal. The left external auditory canal was partially filled with dark brown crusted material which appeared to be old blood. Upon removing this the tympanic membrane was seen to be blue-black and intact. The Eustachian tube was inflated with ease, and no fluid could be heard, nor could bubbles be seen. Audiogram demonstrated a relatively flat conductive hearing loss of 60 db. Roentgenograms showed a densely clouded mastoid in which cellular detail could not be made out. There was a suggestion of bone erosion in the squamosal area.

On Feb. 10, 1953, a mastoidectomy was performed. A bluish-black material of soft consistency filled the entire mastoid cavity, extending into zygomatic and squamosal cells. The bone of the cellular mastoid was softer than normal. When it was found that this same bluish-black material filled the attic and tympanum, a radical procedure was done.

Microscopic examination of this tissue revealed many cholesterol clefts, surrounded by rather dense fibrous tissue infiltrated with lymphocytes and containing numerous small endothelial lined blood vessels. Foreign body giant cells were also present.

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* Surgery performed by Major Samuel Lee and Colonel Wendell A. Weller.

The post operative course was smooth The cavity was completely lined by squamous epithelium 12 weeks following surgery and there had been a 24 db hearing improvement, to the 36 db level

COMMENTS

It is unlikely that any procedure short of radical mastoidectomy would have cured this patient, due to the large amount of abnormal tissue in the middle ear space The hearing improvement following surgery was gratifying to the patient as well as to us

Case 3 A 21 year-old white man complaining of recurrent pain and deafness in his right ear, was admitted to this hospital in June 1953 He stated that he had never been able to hear with his right ear and as long as he could remember he would have four or five episodes of pain a year lasting from a few days to a few weeks Five years prior to this hospitalization he was told that he had a blue eardrum and since that time has been told on a few occasions that he had dried blood in his ear canal He did not remember any episode of frank discharge Mild symptoms of vasomotor rhinitis had been present for years but had not been particularly bothersome He had been referred to this hospital because of a dark fluid draining from the right Eustachian tube area after attempted catheter inflation

Examination revealed a moderate amount of lymphoid tissue over the walls of the nasopharynx and the fossae of Rosenmueller but no obstruction to normal movement of the torus on swallowing The left tympanic membrane was normal The right tympanic membrane was intact appeared to be moderately retracted and was blue black There was no fluid level Initial audiometric examination (pure tone and speech) revealed to 60 db conductive hearing loss in the right ear and normal hearing in the left ear Roentgenograms revealed a pneumatized but densely clouded right mastoid

All attempts to inflate the right Eustachian tube were unsuccessful and at no time was any fluid seen to escape from the tubal orifice as had been described prior to entry Myringotomy resulted in discharge of a chocolate like material similar to fluid from a liquified hematoma The fluid was under slight pressure and a large quantity was removed by suction Hearing improved to the 28 30 db level following almost every myringotomy and aspiration

A diagnosis of idiopathic hemotympanum was made but because of the report of the referring physician of abnormal discharge from the right Eustachian tube a biopsy was taken from the right fossa of Rosenmueller Histologic examination showed normal adenoid tissue

On July 17 1953 a simple mastoidectomy and wide myringotomy were performed The mastoid was well pneumatized and all cells with the exception of the immediate peri antral cells and antrum were filled with a dark granulation like tissue and chocolate-like fluid Sections of the tissue removed revealed osseous and fibrous tissue with an inflammatory infiltrate in the latter consisting of numerous lymphocytes and polymorphonuclear neutrophiles There was a proliferation of fibroblasts and many hemosiderin granules and cholesterol clefts were present Foreign body giant cells were numerous Aerobic and anaerobic cultures of the fluid and tissue removed failed to show any growth

The post operative course was uneventful and ten days following surgery the myringotomy wound was healed and the drum appeared blue-black. There was no hearing improvement, although the patient stated that the ear would occasionally "click and pop," at which time his hearing would improve momentarily. Eustachian tube inflation, however, was unsuccessful.

By the middle of September, although there had been no change in the hearing, there was a change in the eardrum. It took on a gun-metal blue color similar to that seen in serous otitis media, and the fluid became more serous, with much less coloring. It was felt that treatment, aimed primarily at opening the Eustachian tube, would perhaps cure the condition.

A re-adenoidectomy was done, followed in four weeks by a fractionated course of radiation in the region of the nasopharynx, delivering a total of 450 *r* in air to both right and left nasopharyngeal ports over a two-week period.

A month following the completion of X-ray therapy the hearing was at a 32 db level, although there was still serous fluid in the middle ear and a gun-metal blue eardum. No myringotomies had been done in more than a month; however, in the following month the recurrent otalgia returned, and the hearing dropped to a 50 db level.

Based on the experience with Case 2, at this point it was decided that radical mastoidectomy was indicated to rid the patient of his recurrent pain, which was more of a disability to him than the hearing loss. The establishment of a permanent perforation in the tympanic membrane was entertained as a more conservative procedure and one that could possibly result in better hearing than following a radical operation.

On Jan. 21, 1954, under general anesthesia the lower half of the tympanic membrane was turned up, together with the annulus and a few millimeters of skin of the external canal. The middle ear was normal, including the tubal area. A wedged-shaped section of tympanic membrane, including the annulus at the base, was removed, skeletonizing the tip of the manubrium. This represented approximately one quarter of the pars tensa, in the posterior inferior quadrant. Post-operatively a mild otitis media and a bothersome external otitis developed. Both subsided within a week. An audiogram done six days following surgery showed a 23 db hearing loss. Unfortunately this level was never reached again.

No attempt was made to maintain the opening of the perforation, for this was felt to be unnecessary. As healing took place the anterior and posterior edge of the perforation were seen to be on a different plane, the anterior edge being about one to two mm. medial to the posterior edge. There was very little discharge after the first two weeks. Four weeks following surgery the perforation had healed, and the hearing had dropped to 50 db. Again recurrent pain became a complaint, and the eardrum took on a gun-metal blue color. Serous fluid could be aspirated through a myringotomy incision.

On March 18 a further attempt at permanent perforation was made. No evidence remained of the operation just two months before. One third of the pars tensa was removed, the tympanic sulcus was curetted thoroughly, and a selvedge edge gauze pack was placed in the tympanum and external canal to maintain the opening. Wide spectrum antibiotics were prescribed in large doses to prevent a recurrence of otitis media and externa. One week following surgery the gauze pack was removed. The opening was large, the ear was dry, and the hearing was at a 28 db level. Within five days the opening had decreased greatly in size. To prevent closure a small polyethylene tube was placed through the opening into the middle ear and was left in place one month. During this time there were no apparent reactions to the tube. There was no discharge, only mild intermittent pain, and the hearing remained near the 30 db level. When the tube was removed there was a small opening near the floor of the canal, without crusting. Within 24 hours the tympanic membrane had healed, and the hearing dropped to a 40 db level.

Further treatment in the form of a radical mastoidectomy was offered the patient, but was declined for the present.

COMMENTS.

Although this was a typical case of idiopathic hemotympanum, following simple mastoidectomy, it took on the appearance of serous otitis media. It is possible that both conditions existed, but that the serous otitis was masked by the hemotympanum, and that the simple mastoidectomy actually cured the latter. Persistent complete blockage of the Eustachian tube is not common in serous otitis, and the relationship of this to these two conditions is not clear.

Permanent perforation of the tympanic membrane has been used in some resistant cases of serous otitis media with good results,[12] and was suggested to the authors in this case. Immediately following this procedure the hearing was excellent, and, had the opening remained, a permanent improvement could have been expected. Considering the difficulty encountered in attempting to close some perforations associated with a quiescent chronic otorrhea, it is hard to explain why both attempts at permanent perforation failed here. This is particularly true in light of the fact that both were made marginally by removing a portion of the annulus tympanicus.

SUMMARY.

The literature on idiopathic hemotympanum is reviewed. A brief discussion of the clinical course, differential diagnosis, treatment and etiology is presented, along with a detailed description of three cases. The first case was cured by simple mastoidectomy. The second case was treated by radical mastoidectomy, resulting in a cure, but with a conductive hearing loss, less severe than before surgery. The third case was treated unsuccessfully by simple mastoidectomy and two attempts to produce a permanent tympanic perforation.

Since this manuscript was prepared, another case[13] of idiopathic hemotympanum has been reported. Radical mastoidectomy failed to reveal the etiology of the condition, and failed to relieve the patient's otalgia.

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INTERNATIONAL VOICE CONFERENCE.

Following the International Congress of Otolaryngology in Washington, D. C., next Spring, there will be an International Voice Conference (Laryngeal Research, Function and Therapy) in Chicago, Illinois, May 20-22, 1957. For Information address: Dr. Hans von Leden, 30 North Michigan, Chicago 2, Ill., U.S.A.

THE SIGNIFICANCE AND FATE OF THE MESENCHYME OF THE MIDDLE EAR.[*]

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In man, resorption of tympanal mesenchyme begins at the third fetal month and should, ideally, be complete by the eighth. In actuality, traces of this tissue may fairly often be present up to the fourth or fifth year, and a few children retain even larger amounts until the onset of puberty when, under the influence of hormones, any mesenchymal remnants tend to differentiate into adult fibrous tissue The middle ear seems to be one of the last outposts where primitive mesenchyme as an organized tissue (syncytial reticulum) may be found in the developing individual. Pluripotential mesenchyme cells still exist, to be sure, in all the connective tissues and throughout the reticulo-endothelial system, but the masses of syncytium so characteristic of the early embryo are no longer elsewhere in evidence. That the ear should behave in this fashion is perhaps not so strange when we recall that the head of the young embryo consists largely of a mass of primitive mesenchyme containing a brain. The role of mesenchyme, so vast in embryogenesis as a whole, is, therefore, the more striking in the development of the head segment.

Failure of resorption of mesenchyme in the middle ear of man may quite possibly be a hereditary characteristic, and one conceivably associated with some subtle endocrinopathy or metabolic disturbance.[1] It has been pointed[1] out that this fail-

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[*] Read in part at the meeting of the American Association of Anatomists, Philadelphia, Pa , April, 1955.

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[†] This research is being conducted in the Department of Histology of Creighton University under a grant from The National Institute of Health

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suggested the experiment of performing unilateral tubal dilatation in cases of symmetric conduction impairment. This theoretically elegant idea presents one very practical difficulty. Whereas the failure of a few cases to achieve restitution of normal hearing after direct adenoidectomy *without any tubal dilatation* automatically selects a population in which one is entitled to make at least a presumptive diagnosis of unresorbed tubo-tympanal mesenchyme, this selectivity would not operate in so efficient a manner in the procedure suggested by Goodhill. The presumptive mesenchyme-bearing population would not be defined at the time of performance of the crucial procedure, and the requirement of initially symmetrical audiometric curves would drastically limit the sample from which this population would have to be drawn. Since the incidence of unresorbed mesenchyme in children of various ages has never been determined by routine temporal bone studies on general pediatric autopsy material (a study long overdue), accumulation of a fairly extensive series of satisfactory symmetric improvements would not necessarily prove the worthlessness of the unilateral dilatation; nor would its value be easy to estimate, given a few suggestive asymmetric results. Nevertheless, such a long term project should be carried out by someone with adequate clinical facilities in a large pediatric hearing clinic. This investigator should also undertake the tedious but highly necessary study of the incidence of unresorbed mesenchyme in children dying from *all* causes; for not only are otologic deaths today excessively rare, but even had an extensive statistical study of this kind been undertaken on otologic cases in the pre-chemotherapy era, an exaggerated picture of overall mesenchyme incidence would undoubtedly have resulted.

With regard to the safety of the tubal dilatation, Guild[7] has expressed some concern lest the pressure exerted by the olive damage the mucosa of the orifice, especially its delicate ciliary mechanism. It would not be difficult, nor in all probability harmful to biopsy the mucosa after performance of the dilatation; but it seems a scarcely permissible procedure to investigate an alleged possible injury to a patient by inflicting a certainly greater one. We do feel, after eight years' experience with the method, that it is a perfectly safe one. This impression is based in part upon an admittedly crude surgical estimate of the effect upon the tissues of the quite moderate pressure employed, but more particularly upon the absence of any significantly deleterious effects recorded in our follow-up studies. One of us (P. G.)[8] reported rare instances of postoperative otitis media or serous effusion which responded readily to the usual treatments with no impairment whatever of the ultimate results. It is also worth mentioning that such complications will occasionally occur also after adenoidectomy *without* tubal dilatation.

In view of the widespread popularity of radiation treatment of adenoid remnants (and even of unremoved adenoids) which prevailed from 1941 until recently, it is rather surprising that no consideration has been given to the possible effects of ionizing radiation upon unresorbed tubo-tympanal mesenchyme. Conceivably such radiation could promote either resorption or differentiation into scar tissue. This problem is presently under experimental investigation in our laboratory.

It is readily apparent, in view of the paucity of both clinical and pathological information regarding unresorbed tubo-tympanal mesenchyme in man, that animal experimentation is needed. Moses Lurie,[9] of Harvard University, was the first to attempt this. Prior to 1936 he studied a strain of dwarf mice in which he found mesenchyme to be retained in the tympanic cavity indefinitely unless the animals were treated by injections of pituitary extract. L. K. Guggenheim[1] attempted to carry out similar experiments but with inconclusive results.

Stimulated by the work of these two men we have attempted to study the normal development of the middle ear of the white rat with reference especially to the cycle of growth and decay of the mesenchyme. It appears that this post natal study in the rat would correspond to an embryologic study in man because of the difference in the stage of the mesenchyme cycle of growth and resorption with respect to the event of birth. It seems likely, however, that much the same factors must be responsible for the behavior of this tissue in both rat and man. It is intended that the base-line established by this study shall be a standard against which to measure the effects of various chemical and physical agents which might experimentally influence the mesenchyme.

suggested the experiment of performing unilateral tubal dilatation in cases of symmetric conduction impairment. This theoretically elegant idea presents one very practical difficulty. Whereas the failure of a few cases to achieve restitution of normal hearing after direct adenoidectomy *without any tubal dilatation* automatically selects a population in which one is entitled to make at least a presumptive diagnosis of unresorbed tubo-tympanal mesenchyme, this selectivity would not operate in so efficient a manner in the procedure suggested by Goodhill. The presumptive mesenchyme-bearing population would not be defined at the time of performance of the crucial procedure, and the requirement of initially symmetrical audiometric curves would drastically limit the sample from which this population would have to be drawn. Since the incidence of unresorbed mesenchyme in children of various ages has never been determined by routine temporal bone studies on general pediatric autopsy material (a study long overdue), accumulation of a fairly extensive series of satisfactory symmetric improvements would not necessarily prove the worthlessness of the unilateral dilatation; nor would its value be easy to estimate, given a few suggestive asymmetric results. Nevertheless, such a long term project should be carried out by someone with adequate clinical facilities in a large pediatric hearing clinic. This investigator should also undertake the tedious but highly necessary study of the incidence of unresorbed mesenchyme in children dying from *all* causes; for not only are otologic deaths today excessively rare, but even had an extensive statistical study of this kind been undertaken on otologic cases in the pre-chemotherapy era, an exaggerated picture of overall mesenchyme incidence would undoubtedly have resulted.

With regard to the safety of the tubal dilatation, Guild[7] has expressed some concern lest the pressure exerted by the olive damage the mucosa of the orifice, especially its delicate ciliary mechanism. It would not be difficult, nor in all probability harmful to biopsy the mucosa after performance of the dilatation; but it seems a scarcely permissible procedure to investigate an alleged possible injury to a patient by inflicting a certainly greater one. We do feel, after eight years' experience with the method, that it is a perfectly safe one. This impression is based in part upon an admittedly crude surgical estimate of the effect upon the tissues of the quite moderate pressure employed, but more particularly upon the absence of any significantly deleterious effects recorded in our follow-up studies. One of us (P. G.)[3] reported rare instances of postoperative otitis media or serous effusion which responded readily to the usual treatments with no impairment whatever of the ultimate results. It is also worth mentioning that such complications will occasionally occur also after adenoidectomy *without* tubal dilatation.

In view of the widespread popularity of radiation treatment of adenoid remnants (and even of unremoved adenoids) which prevailed from 1941 until recently, it is rather surprising that no consideration has been given to the possible effects of ionizing radiation upon unresorbed tubo-tympanal mesenchyme. Conceivably such radiation could promote either resorption or differentiation into scar tissue. This problem [illegible] presently under experimental investigation in our laboratory.

It is readily apparent, in view of the [illegible] clinical and pathological information regarding [illegible] tympanal mesenchyme in man, that animal experimentation is needed. Moses Lurie,[5] of Harvard University, was the first to attempt this. Prior to 1936 he studied a strain of dwarf m[illegible] which he found mesenchyme to be retained in the tympanic cavity indefinitely unless the animals were treated by injections of pituitary extract. L. K. Guggenheim[4] attempted to carry out similar experiments but with inconclusive results.

Stimulated by the work of these two men we have attempted to study the normal development of the middle ear of the white rat with reference especially to the cycle of growth and decay of the mesenchyme. It appears that this post natal study in the rat would correspond to an embryologic study in man because of the difference in the stage of the mesenchyme cycle of growth and resorption with respect to the event of birth. It seems likely, however, that much the same factors must be responsible for the behavior of this tissue in both rat and man. It is intended that the base-line established by this study shall be a standard against which to measure the effects of various chemical and physical agents which might in any fashion influence the mesenchyme.

Realizing, too, that Lurie's dwarf mouse provided a most elegant experimental object, we attempted to procure such animals from the same sources. We found that the animals sent us exhibited a cycle of growth and regression of tympanal mesenchyme quite similar to that observed in our study of the white rat. Specimens from other widely scattered sources also revealed no instance of indefinite retention of mesenchyme. It seemed clear that Lurie had been so fortunate as to have obtained a rare mutant which is now either extinct or unavailable. The systematic study of the tympanal mesenchyme cycle of dwarf mice and their normal heterozygous littermates is presently being made and will be reported subsequently. The present report will be limited to results with the white rat.

Some comparisons between the ears of rat and man are useful for purposes of orientation in embryologic time. Ossification of the labyrinthine capsule of man first begins at the end of three months gestation, but is accomplished principally during the fifth month. The earliest deposition of calcium occurs in the matrix of the cartilage, most of which is subsequently removed by invading osteogenic buds. The remaining calicfied matrix is converted into intrachondral bone when the swollen lacunae are invaded by osteogenic buds which proceed to lay down bone within them. Anson and Bast[10] speak of the cartilagenous otic capsule as being "almost completely converted into a bony capsule by the 202-mm. stage (23rd week)," but state that it consists at this stage of islands of intrachondral bone lying in large primitive marrow spaces. These spaces are only very slowly diminished by deposition of endochondral bone, a process which becomes greatly accelerated just before birth.

In the rat, as in man, the first evidence of impending bone formation is seen to be a swelling of the cartilage lacunae. This was first noted in the posterior lip of the internal auditory meatus of the rat at three days after birth. By five days extensive calcification has occurred on the medial side of the cochlear capsule, including the modiolus. The promontorial wall forming the lateral boundary of the second cochlear turn shows calcification (site of attachment of ligamentum spiralis). Centers are noted on the anterior lip of the internal auditory meatus and on the medial aspect of the double ampulla of the vertical canals. The cochlear promontory and medial tympanic wall posterior to the oval window now show swelling of cartilage lacunae. By nine days, almost the entire cochlear capsule has been converted, and beginning changes are occurring in the various parts of the vestibular labyrinth. At this stage the bony labyrinth consists of a tenuous framework of intrachondral bone lying amidst enormous marrow spaces. In consequence, it has a most fragile appearance and is still capable of being sectioned by a sliding microtome without decalcification. From about ten days on, one can notice the earliest harbinger of endochondral bone formation in the form of thin lamellae of ossein matrix around the islands of intrachondral bone. From this time forth the capsule assumes a progressively more substantial appearance as the marrow spaces diminish in size. At 11 days the vestibular capsule is extensively converted. *A very rough estimate might equate a rat of five days with a human fetus of five months.*

With regard to the development of the cochlea, the classic work of Wada[11] states that the rat hears clearly on the 12th day of life, sometimes as early as the 10th or 11th day. (One instance was recorded in which hearing occurred on the ninth day.) The 12th day of the rat would, therefore, be comparable to the end of the sixth fetal month in man, when the organ of Corti is fully developed and physiologically competent. The week from the fifth to the 12th day in the rat thus *corresponds roughly to the sixth month of gestation in man.*

Wada's monograph[11] nowhere mentions mesenchyme, but we have had the opportunity of studying his original collection of slides at the Wistar Institute (being the second visitors to disturb them in the last quarter of a century). His sections are cut in a radial-vertical plane in order best to exhibit the cochlea. Ears were sectioned at three-day intervals from birth. An insufficient number of sections of each specimen was represented to tell the whole mesenchyme story, inasmuch as changes are occurring with a rapidity which demands specimens taken at daily intervals. The picture of development reconstructed from this series coincides very closely with that derived from our own.

Certain anatomic peculiarities of the rat are likely to confuse the otolaryngologist used to thinking in human terms. The nose adjoins the nasopharynx by a quite novel anatomic arrangement, which has been studied in detail by Kelemen.[12] The nasopharynx itself differs strikingly in configuration from man, being a long funnel-like structure which may be traced caudad in serial sections until it almost literally drops into the orifice of the larynx, the communication between it and the oral cavity being very small indeed.

No vestiges whatever of either tonsils or adenoids were seen in any of the animals studied by us, which were standard laboratory rats of the Sprague-Dawley and Holzman strains. It may well be that pharyngeal lymphoid tissue appears later in the animal's development than we followed it for our purposes. According to Greene,[13] rats, unlike other familiar laboratory animals, lack tonsils except as scattered lymphoid follicles. She makes no mention of adenoids.

TECHNICAL PROCEDURE AND HISTOLOGIC FINDINGS.

Rats were sacrificed at daily intervals from one to 27 days of age by transaortic injection of Zenker's solution or formalin under general anesthesia with intraperitoneal nembutal.* The internal jugular veins were severed in order to facilitate perfusion of the head. The animals were decapitated immediately postmortem, heads skinned, calvaria and brains removed, and the mandibles excised. The remains were further fixed by immersion in fixing fluid for 12 to 24 hours. Specimens from 11 days on were decalcified in three per cent nitric acid solution for 48 hours. Celloidin was used for embedding. Sections were hand cut by sliding microtome in a plane parallel to the hard palate. Those not decalcified were sectioned at 10μ thickness. The decalcified heads were sectioned at gradually increasing thicknesses, until, from 15 days on, all sections were of 20μ thickness. Sections were stained in rotating order with iron-hematoxylin, hematoxylin-eosin-azure, orcein, silver nitrate, and Mallory's connective tissue stain.

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*Intravital fixation by injection under anesthesia was first employed by Siebenmann & Yoshii (1908), Metzner & Yoshii (1909) and Sato (1917), as mentioned by Wada (op. cit.) Yoshii used ether anesthesia and injected transaortically a solution consisting of: formol 10 per cent, 74 parts; Muellers fluid, 24 parts; glacial acetic acid, 2 parts.

In addition to the recording of descriptive data, an effort was made to study more precisely the growth and decay of the mesenchyme mass in relation to the overall growth of the potential tympanic cavity. Slides were projected onto standard white typing paper of known weight per unit area at 38x magnification, and the tracings cut out and weighed. From the paperweight and magnification data, actual areas on the slides were calculated. For purposes of convenience, the tympanum was divided into two regions, arbitrarily designated "Tubo-promontorial" (TP) and "Stapedial foot-plate" (S). Insofar as possible, the TP region was represented by a section which showed the full length of the Eustachian tube from nasopharynx to tympanum. The posterior boundary of this region, for purposes of quantitative study, was arbitrarily taken as the malleus. The S region was represented by a section showing the juncture of the anterior stapedial crus with the footplate. The S region thus lies cephalad to the TP region. Sections from the TP region of very young animals* show not only a corridor of free space lateral to the promontorial mesenchyme pad, but also a somewhat smaller chink extending just medial to the malleus and then posteriorly for almost the full length of the potential cavity (see Fig. 1). This posterior chink is evident only in sections caudal to the stapes

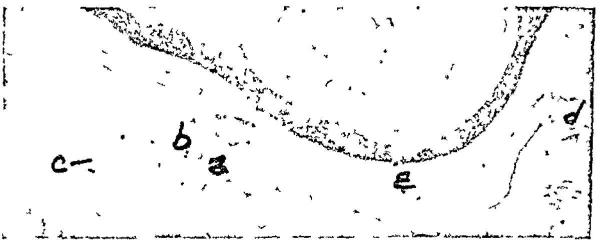

FIG. 1 Rat one day old Composite photomicrograph showing entire extent of actual tympanic cavity at TP level

a Free tympanic lumen, b Malleus, c Drum membrane, d Tympanic orifice of Eustachian tube, e Anterior promontorial mesenchyme pad footplate, the S region being solidly packed with mesenchyme (see Fig. 2). Due to the arbitrary selection of levels for quantitative study, this extension of free space posterior to the malleus has not received systematic quantitative consideration.

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* We have studied specimens as early as one day prenatal An embryologic series is now in preparation

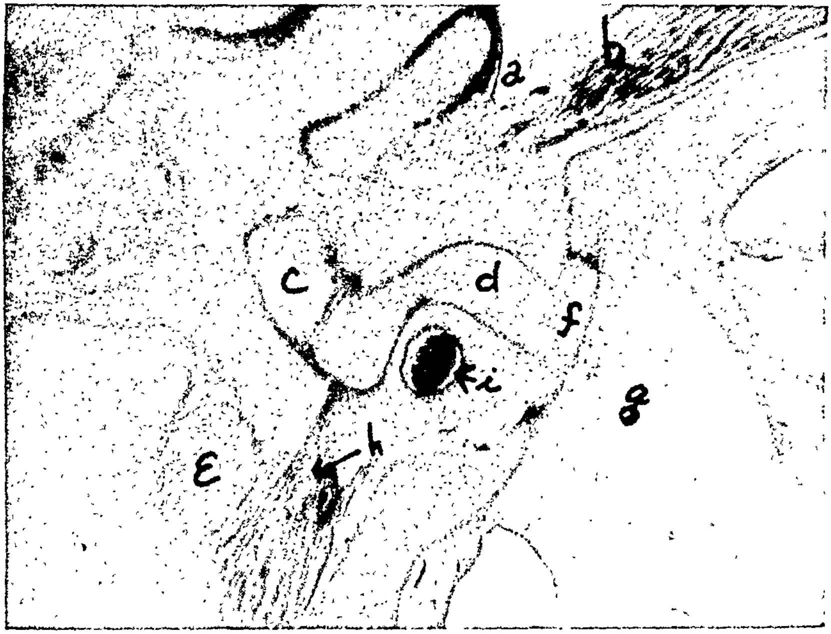


Fig. 2. Rat at one day. Transverse section at stapes footplate level (S). a. Comma of free space extending just medial to malleus but not further posterity at this more cephalic level (cf. Fig. 1); b. Tensor tympani muscle lying just lateral to cochlear promontory; c. Incus; d. Anterior crus of stapes; e. *Nervus facialis*; f. *Stapedial footplate*; g. *Labyrinthine* vestibule; h. Stapedial muscle; i. Stapedial artery.

Table I shows actual cross-section areas of mesenchyme in relation to areas for the whole potential tympanic cavity in the S region for animals of various ages.

Note that all specimens through the 11th day show a progressive increase in the size of the mesenchyme mass exactly paralleling the growth of the potential tympanic cavity at this level (S). After the 11th day, the mesenchyme begins to decline in the absolute amount while the S region of the potential cavity continues to grow very slowly. The amount of free space gradually increases until by 20 days there is almost total elimination of mesenchyme.

TABLE I.

Stage (days)	Area of Potential Tympanic Cavity (mm.^{2})	Area of Mesenchyme (mm.^{2})	Area of Free Space (mm.^{2})
1	3.4	3.4	0.0
2	4.6	4.6	0.0
3	6.6	6.6	0.0
5	6.6	6.6	0.0
7	6.9	6.9	0.0
9	7.2	7.2	0.0
11	7.5	7.5	0.0
12	7.8	6.9	0.9
13	8.2	5.7	2.5
15	10.2	3.3	6.9
16	10.2	1.2	9.0
17	9.7	1.3	8.4
19	10.0	1.2	8.8
20	10.3	1.2	9.1

Stapedial Footplate Region (S).

TABLE II.

Stage	Area of Potential Tympanic Cavity (mm.^{2})	Area of Mesenchyme (mm.^{2})	Area of Free Space (mm.^{2})
1	2.5	0.6	1.9
2	3.4	2.5	0.9
3	4.0	2.5	1.5
5	4.0	2.8	1.2
7	8.5	3.9	4.6
9	9.5	3.4	6.1
11	17.0	5.4	11.6
13	20.0	0.4	19.6
15	21.5	0.0	21.5
16	22.0	0.0	22.0

Tubo-promontorial Region (TP).

Table II shows the same data for the TP region.

Here it will be noted that the trends are, in general, the same as in the S region, but certain differences are striking. The mesenchyme in the TP region does not fill the entire cavity on the first day, and figures for the mesenchyme and total cavity areas begin to diverge more rapidly than in the S region. As in the S region, the mesenchymal mass increases in absolute amount until the 11th day, when a maximum is reached. From the 11th to the 13th day there is a slow decline which becomes precipitous after the 13th day. The more advanced cavitation in the TP region is due primarily to the more rapid growth of this part of the potential cavity. It is obvious, too, that the resorption of mesenchyme proceeds more rapidly in the TP region, being complete at 15 days, whereas in the S region, remnants may linger up to 23 days. These changes are represented in the accompanying graph (see Fig. 3) and illustrated by the photomicrographs (see Figs. 1 and 2, and Figs. 4 to 10).

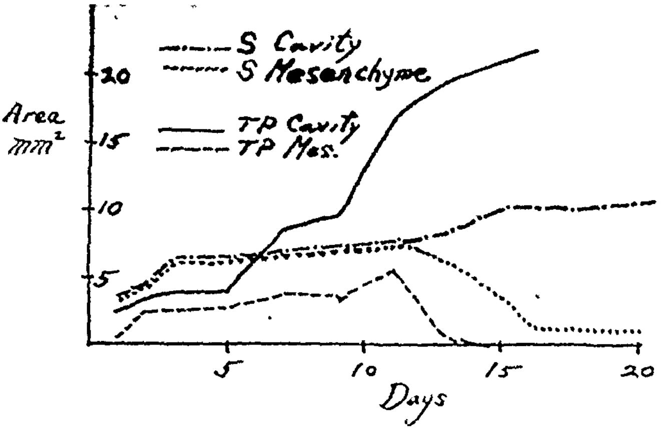


Fig. 3. Graphical representation of data in Tables I and II.

The foregoing study tells us nothing about the mechanism

Fig. 4. Rat at nine days. TP region.

a. Approach to the tympanic orifice of Eustachian tube (a'); b. Anterior promontorial mesenchyme polyp—Note greater density of ground substance, as indicated by more homogeneous, dark-staining appearance compared with c. mesenchyme of posterior part of tympanum which, as yet, shows no change preparatory to resorption; d. Malleus; e. Drum membrane.

Fig. 5. Rat at 12 days. TP region under higher magnification.

a. Tympanic orifice of Eustachian tube; b. Anterior promontorial polyp showing web-like structure with large holes, indicating impending dissolution.

Fig. 6. Rat at 15 days. TP region. Note complete absence of mesenchyme at this level.

a. Tympanic orifice of Eustachian tube.

Fig. 7. Rat at 17 days. S region.

a. Tensor tympani muscle; b. Anterior crus of stapes; c. Posterior crus of stapes; d. Footplate of stapes; e. Stapedial artery; f. Facial nerve; g. Free tympanic lumen (g', g''—holes in the mesenchyme around the stapes); h. Vestibule of the labyrinth; i. Stapedius muscle.

Note that various components of stapes appear to be composed of intrachondral bone

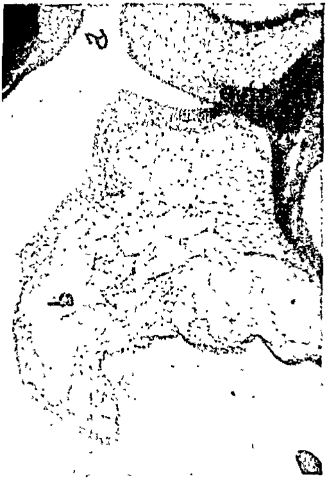
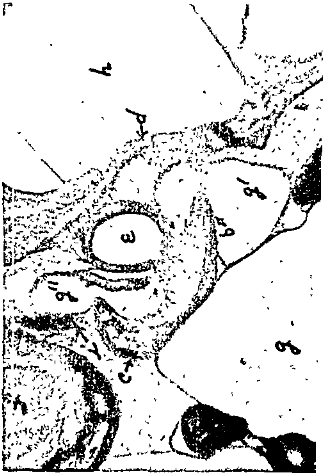


Fig. 3



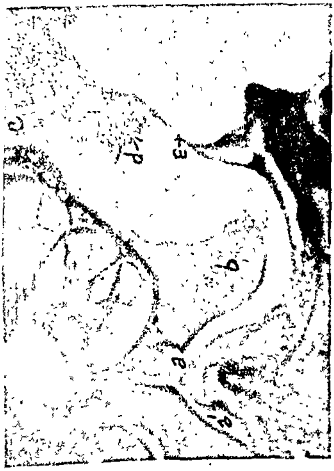
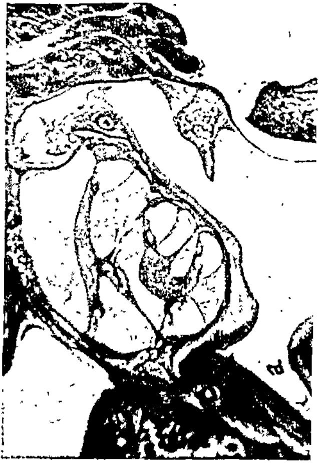


Fig. 4



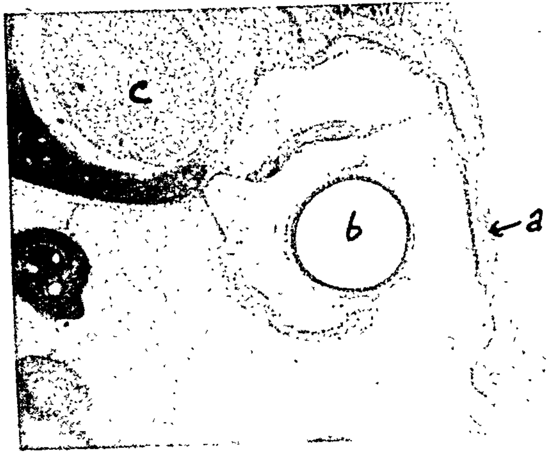


Fig. 10.

Fig. 8. Rat at one day. Wada collection,* vertical section, showing stapedial footplate.

a. Free tympanic lumen; b., c. Malleus, incus; d. Stapedial artery; e. Facial nerve; f. Part of the footplate derived from Reichert's ring —represented by darker-staining kidney-shaped area—flattened on its medial surface to accommodate itself to f.' part of the footplate derived from curtain of the oval window (labyrinthine capsule proper);[14] g. Anlage of annular ligament; h. Vestibule of labyrinth; i. cochlea.

Fig. 9. Rat at 12 days. Wada collection.* Footplate region. Vertical section.

a. Free tympanic lumen; b., c. Malleus, incus; d. Stapedial artery; e. Stapes footplate, now composed of intrachondral bone; f. Facial nerve.

Fig. 10. Rat 25 days. Wada collection,* vertical section, footplate region. No trace of mesenchyme remains in the stapedial region.

a. Footplate of stapes; b. Stapedial artery; c. Facial nerve.

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* These photomicrographs were made at the Creighton University with permission of the Wistar Institute.

of dissolution of the tympanal mesenchyme mass—a most intriguing problem. A similar mesenchymal dissolution occurs in the formation of the perilymph chambers (at two months in man)[15] and of the subarachnoid space,[16] with the difference that the spaces vacated by the mesenchyme in these instances come to be occupied respectively by the homologous perilymph and cerebrospinal fluids, instead of by air as in the tympanic cavity. It may well be that a "liquefaction" occurs here also, the fluid being drained off through the Eustachian tube, or perhaps partly absorbed by adjacent lymphatics or capillaries.

The middle ear of the rat is one of the few locations where a mass of primitive mesenchyme is destined to disappear entirely after birth, and one which lends itself readily to observation. Otologists have termed the mechanism of this disappearance "resorption." Strictly speaking, the term "resorption" implies incorporation of the material "resorbed" into the structures of its environment. Undoubtedly some of the tympanic mesenchyme cells contribute to the elaboration of other structures in the potential tympanic cavity (especially those cells which are destined to become periosteum), but the vast majority simply disappear. "Dissolution" or "disintegration" would, therefore, seem better terms here than "resorption." The latter, however, is so entrenched in otologic literature as to be virtually invulnerable to such considerations. Obviously neither term could possibly be expected to epitomize so complex a process.

One cannot help being struck by a resemblance between the last remaining shreds of *moth-eaten* mesenchyme in the middle ear (see Fig. 7) and the trabeculae of the subarachnoid and perilymphatic spaces. Whereas these trabeculae have a most necessary supportive function where normally found, homologous remnants in the tympanic cavity represent an *arrest* of normal development and result in impaired mobility of the ossicular mechanism.

Schwarz[17] in 1931 spoke of "pneumatization" of the tympanic mesenchyme, claiming that this process begins earlier in the human fetus (three months) than previously supposed. The term "pneumatization" at once suggests to the otologic mind the mechanism of formation of the mastoid air cells.

into histiocytes and vacuolated cells was carried out for both TP and S regions. It was noted that these "differentiated" cells existed in unevenly distributed clusters. Hence selection of areas for cell counts involved a highly subjective choice factor. For this reason, a small area was chosen in the S region which showed a great stability in size and configuration throughout the series of animals studied: an area bounded by the stapedial artery, footplate and anterior crus. The increase in the two types of so-called "differentiated" cells mentioned above as a function of age is shown in Table V.

TABLE V.

Age of Animal in Days	Macrophages	Vacuolated Cells
1	0	0
2	0	0
3	0	0
5	0	0
7	1	1
9	5	2
11	7	7
12	7	8
13	11	6
15	11	8
17	10	9
18	20	10

Numbers of differentiated cells as a function of age. Area bounded by anterior stapedial crus, stapedial artery and footplate.

The increase in the number of differentiated cells as the age of the animals increases is quite apparent. A comparison with Table III, which lists cell densities for this region, indicates that the increase of differentiated cells occurs at the same time that the total number of cells is decreasing. In the older stages noted in Table V, practically all the cells show some degree of differentiation.

The corresponding study in the TP region is complicated by the fact that the mesenchyme here is considerably less homogeneous. Three layers are apparent from the first day of life and become increasingly distinct with advancing development:

1 The deepest layer, next to the perichondrium* (later, periosteum) of the promontory,

2. The central layer, which constitutes the bulk of the total mass, and

3 The layer adjacent to the free luminal border of the mass

Layers 1 and 3 appear not only to have a more condensed intercellular substance than Layer 2, but a higher total cell population and a higher incidence of differentiated cells These data are shown in Table VI

TABLE VI

Age Animal In Days	Layer 1			Layer 2			Layer 3		
	(a)	(b)	(c)	(a)	(b)	(c)	(a)	(b)	(c)
1	25	4	0	11	1	1	21	0	0
2	11	0	0	7	1	0	18	2	0
3	20	0	0	8	1	0	22	7	0
[illegible]	9	0	0	3	0	0	20	6	0
[illegible]	10	0	1	3	1	0	12	3	0
9	10	4	0	5	0	0	7	3	0
11	5	1	0	1	0	1	4	2	1
12	32	3	6	3	0	0	42	12	8
13	0	0	0	0	0	0	0	0	0

Changes in three layers of the promontorial mesenchyme pad
Layer 1 is the layer next to the perichondrium periosteum of the cochlear promontory
Layer 2 is the large, inert central portion
Layer 3 is the layer next to the free tympanic lumen
(a) Total cell population
(b) Histiocytic cell population
(c) Vacuolated cell population

It would appear that the large and apparently inert middle layer of the promontorial mesenchyme pad disintegrates with the least exhibition of "differentiated cells" Consequently the formation of histiocytes and signet ring cells can hardly be the principal mechanism of disposal of this layer It may well be that an enzymatic liquefaction of the stroma occurs, presumably involving a depolymerization of the ground substance

Converse changes in the ground substance of the *actively*

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* A very thin perichondrium is easily distinguishable on the first day of life At 11 days (see Fig 12) this layer is notably thickened Undoubtedly mesenchymal cells are here becoming periosteal fibroblasts For purposes of this quantitative study the perichondrial periosteal layer is excluded from the mesenchyme mass as now constituting a definite structure separate from it

growing mesenchyme of placental villae were studied by Gersh and Catchpole.[18] Of special interest was the finding of intracellular glycoprotein granules which reached a maximum population density at four months, corresponding to the time of maximum blood and urinary content of chorionic gonadotropin. The fetal placental mesenchyme cell was thought perhaps to be a specific target of this hormone. The number of granules declined from four months to term, when only rare cells exhibited them.

The significance of the vacuolated and signet ring cells was not at first apparent since fat, as such, is not normally found in the tympanic cavity. (Dorothy Wolff[2] described fat in the sinus tympani of two autopsied children, ages 13 months and eight years). The appearance of fat droplets in mesenchyme cells was apparently first described by Lewis[19] in 1918. She explanted 6 to 9 day chick embryos into nutrient medium containing Sudan III, and observed that "the fat droplets of the mesenchyme cells remain distinctly smaller than those of the clasmocyte." (Her purpose appears to have been to show that these droplets did *not* arise as a result of mitochondrial differentiation.), It goes without saying that Sudanophilia is not today considered conclusive evidence of the lipoid nature of an intracellular inclusion. Even the signet ring cells observed by us, which were first described in mesenchyme as early as 1925 by Ernst,[20] cannot simply be assumed to be fat cells. Further histochemical investigation is now underway in an effort to settle this question. Pending results of this study, we have received considerable enlightenment from the work of Holtfreter[21,22,23] on the transformation of lipochondria into the larger liposomes by enzymatic proteolysis of their surface films, a process to which he has given the name "lipophaneresis." The appearance of larger cytoplasmic inclusions of unmasked fat due to coalescence of smaller inclusions was described by Bungenberg De Jong,[24] who called it "coacervation." Holtfreter[22] states that the appearance of fat droplets, vacuoles and granules is common in physically irritated cells, *e.g.*, in ageing tissue cultures. Their presence is pathological, but does not necessarily lead directly to the death of the cell. The unmasking of lipids is apparently due to liberation of an enzyme normally inactive because combined with other cytoplasmic compounds. These considerations lend weight to the hypothesis that mesenchyme dissolution in the middle ear is an enzymatically determined process, and a mechanism is suggested by means of which the dilatation of the tubal orifices with the Gyergyai olives might be effective: liberation, by compression, of a proteolytic enzyme either from the epithelium of the dilated region or from submucosal remnants of unresorbed mesenchyme beneath it. Further work, it is hoped, will put this hypothesis to a test.

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POST GRADUATE COURSES AT TEMPLE UNIVERSITY

Postgraduate Course in Bronchoesophagology, February 11th - 22nd, 1957 — May 20th - 31st, 1957 — September 9th - 20th, 1957.

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Further information and application blanks can be obtained from Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia, 40, Penna.

ODONTOPATHIC ASPECTS IN OTOLARYNGOLOGY*†

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Many patients present themselves to the otolaryngologist with problems that have as their source dental structures. It is these problems that will be discussed, especially in regard to diagnosis and complications.

PAIN OF DENTAL ORIGIN.

Facial Pain — Facial pain often presents a perplexing problem, not only to the patient, but also to the otolaryngologist. He should be able to evaluate dental structures to some degree and should be aware that the teeth may be the etiology in pain about the face.

Inflammation of the internal soft tissues of the teeth, known as pulpitis, may be the source of vague facial pain. The patient sometimes is unable to localize an offending tooth. Usually pain referred to the ear comes from a mandibular tooth; pain in the eye or upper part of the face has as its source, an upper tooth. This is not an absolute rule, and in an occasional instance pain may differ entirely from this pattern. The patient may complain of what he calls "headache" about the temporal and facial regions. Since the dental pulp responds to irritation by inflammation, it is necessary in examination to note, *1.* whether caries are present; *2.* whether recent dental treatment has been received. Dental restoration may have resulted in inadvertant injury to the pulp through its exposure, excessive heat, or irritation by certain fillings, such as the silicates. Traumatic occlusion by a high spot on

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the filling may cause irritation. The latter group, especially, may be puzzling to the otolaryngologist, since he may see a well restored tooth and not suspect that this tooth with its pulpitis is the source of pain.

The use of thermal stimuli is a great help in diagnosis. The normal pulp pain response to extremes of heat and cold leaves in a few seconds, as soon as the stimulus is removed. In an inflamed pulp the response persists. For measuring fine differences in threshold of response an electric pulp tester is needed.

As the inflammation persists, necrosis takes place, and the patient may be comfortable for varying periods of time (days to months, even years) until the necrotic tissue of the pulp becomes infected with enough virulent organisms to involve the tissues surrounding the tooth, resulting in periapical infection, abscess, osteitis or osteomyelitis. The affected tooth responds to percussion by increased tenderness as compared to percussion of the other teeth. Percussion is an easy and accurate means of localization of periapical involvement. The otolaryngologist can easily use the handle of his laryngeal mirror to tap several teeth in evaluating gross differences. Since the nerve tissue of the pulp has died, there is little reaction of the tooth at this stage to thermal changes. The next step is spread of the infection as a cellulitis or formation of an abscess. It might be well to point out that after a periapical abscess has drained into the facial soft tissue from within the bone, percussion tenderness may be absent; but the facial swelling is often characteristic. The parietal abscess that is located lateral to the tooth and is often secondary to periodontal disease may be readily localized by the inflamed surrounding gingiva. It is frequently associated with at least some sensitivity to percussion, although the tenderness to percussion may not be outstanding.

Post Extraction Osteitis — Recent extraction of teeth may present a problem of diagnosis. A patient comes to the otolaryngologist with the history that he has a one-sided facial pain for which a tooth had been extracted with temporary relief; and now having a return of dull aching facial pain, four to five days after the extraction, he seeks another cause. This is the typical story of the "dry socket" or post-extraction osteitis which usually subsides spontaneously within about 14 days. The patient may be given relief and at the same time a therapeutic test, by cleansing of the tooth socket and the use of a paste ointment containing an antiseptic and topical anesthetic. The presence of fever and continual pain may mean extension of infection, and osteomyelitis must be considered.

Unilateral Pharyngitis — The discomfort of a unilateral sore throat or pharyngitis, especially in a young adult, may have as its etiology a pericoronitis or inflammation about a partially erupted third molar. This is usually caused by infection under the remaining flap of tissue, and frequently is associated with a certain amount of trismus. The diagnosis of this condition and its complications (which will be discussed later) is of much importance to the otolaryngologist. Occasionally unilateral pharyngitis may be secondary to dental treatment. Inflammation or hemorrhage within the internal pterygoid muscle may occur as the result of the use of local anesthesia in a mandibular block. Fortunately, infection, broken needles or injection of wrong solutions are now relatively rare complications, even though the use of local anesthesia has increased in restorative dentistry.

Impacted Teeth — An impacted third molar may cause vague and variable discomfort. Impacted teeth in themselves, however, may be completely symptomless. These teeth are often malformed and malpositioned, hidden completely or partially by bone and soft tissue. Thus, they may cause discomfort by pressure on the mandibular or maxillary nerves or on adjacent teeth, even rarely causing exposure of the pulp of the adjacent tooth. Adequate X-ray studies of the third molar area are necessary to note the presence and status of an impacted molar. Other teeth, especially the upper canines, may remain unerupted and hidden to cause facial pain.

Temporomandibular Joint — Pain about the face and ear may be associated with the temporomandibular joint. It is important to remember that this area may be affected with the same diseases and conditions that affect the other joints of the body. The jaw muscles, ligaments and articulating surfaces may be subjected to traumatic, inflammatory and degenerative diseases.

The history, especially for evidence of degenerative and systemic arthritis, and recent or old trauma is important. Knowledge of a recent difficult mandibular dental extraction, surgery in the oropharyngeal regions, especially under general anesthesia, and previous facial fractures involving the mandible are important.

Examination of the joint for subluxation should be tested. Pain may be produced when a finger is placed over the joint as the patient opens his mouth and also moves the mandible laterally. This helps in diagnosis. Frequently the muscles and ligaments are the source of the discomfort rather than the articulating surface. Selective injection of a local anesthetic in various sites, as in the joint and/or its associated muscles will help in localization. X-ray examination should be carried out in the diagnostic workup of temporomandibular joint pathology. Frequently this will be disappointing.

Evaluation of dental occlusion is important, mainly because imbalance of occlusal function leads to traumatic strain of the ligaments, muscles and articulating surfaces of the temporomandibular joint.

Treatment is based on diagnosis and removal of underlying causes. This may include treatment of a psychic tension state in bruxism, establishment of balance occlusion where indicated and/or systemic medication in the infections and degenerative arthritis.

As with any joint disease, joint rest is essential. The patient should be discouraged from experiments with jaw movements to reproduce the pain. Chewing gum, pipe smoking and similar habits should not be permitted. Often the only method of obtaining joint rest is by inter-maxillary wiring. In acute states, injection of a cubic centimeter or more of Hydrocortisone with procaine into the joint is useful. Application of heat aids in decreasing muscle spasm. Salicylates still remain valuable therapeutic agents. The otolaryngologist should be aware that surgical procedures, such as meniscectomy and condylectomy, may become necessary. Rongetti suggests that a rational approach for meniscectomy is through the endaural route. A sclerosing agent injected into the temporomandibular joint in excessive subluxation has been used with some degree of success.

Stomatitis — Stomatitis resulting from various infections and oral manifestations of systemic disease is not within the province of this paper. The otolaryngologist should be aware, however, of the irritations and allergic idiosyncrasies that may be produced by medications, mouth washes, dental plates, dental powders and dentures. Materials containing synthetic detergents and denture plastics that are not completely polymerized (resilient plastics) are the most frequent offenders.

SURGICAL CONSIDERATIONS.

Space Abscess — Let us consider now the space abscess and what it represents clinically. It may be defined for our purposes as a collection of pus within the connective tissues of the face. Numerous types occur, but our discussion is confined to those related to the back part of the jaws. These are the pterygomandibular, infratemporal, parapharyngeal and parotid space abscesses.

An otolaryngologist is familiar with the toxic adult patient who is unable to open his mouth. His first thought is a peritonsillar abscess or peritonsillitis, for these often limit opening. Within a few centimeters of these structures, however, lie the third molars, or wisdom teeth. Infection arising from these may also restrict movement of the mandible and cause severe pain. As stated above, inflammation surrounding the crown of the third molar occurs. This inflammation or pericoronitis, may extend some distance from its source. Its spread into inaccessible areas, where abscess can form, becomes a major complication of pericoronitis.

It should be explained before continuing, that the description of so-called spaces is for simplicity. They are not spaces at all, b
compartm

Pterygomandibular space abscess is the first to be discussed. *It is found between the mandible and inferior part of the* internal pterygoid muscle. A patient may suffer for days, because the doctor cannot open the patient's mouth to see what is happening. A preceding pericoronitis is usually the cause, but sometimes needle borne infection is carried into the space. Aspiration is helpful in establishing a diagnosis before incision is performed. Treatment may be attempted under local anesthesia, but occasionally general anesthesia may be needed to gain access to the mouth.

Infratemporal abscess is the second complication of pericoronal infection. This area lies between the vertical ramus of the mandible and the upper portion of the pterygoid muscle. It is necessary, then, to differentiate carefully since each abscess is incised in a different place. This infection, limited by bone and muscle tissue, does not produce extensive swelling unless the infection spreads toward the temporal fossa externally, or toward the pharynx internally. A thrombophlebitic process of the pterygoid and pharyngeal plexuses must be considered when swelling in the oropharynx occurs. Incision and drainage, usually under local anesthesia, is the definitive treatment when this is possible. In extensive infections a combined internal and external approach may have to be employed.

Parapharyngeal space abscess is a third complication of pericoronitis. The anterior compartment of this space is located between the internal pterygoid muscle and the superior constrictor of the pharynx. A posterior part is also present, but this is not as often involved. As with the others, trismus is again a prominent sign. Severe pain may occur with swallowing, and peritonsillar abscess must be differentiated. Antibiotics are administered liberally as medical treatment is tried, and by the time the patient is seen by the otolaryngologist, the picture has been complicated. Incision and drainage are indicated, but in this area spontaneous drainage or resolution will at times occur.

A fourth infection is simply that between the two connective tissue layers surrounding the parotid gland. Severe pain in the parotid area develops. Differentiation from a parotid tumor is important and not always easily determined, especially in this antibiotic era.

Submaxillary space infection usually occurs as extension from apical abscess of bicuspid and first and second molars. It may also occur, however, from pericoronal infection in third molars.

Abscess in the spaces discussed include the more difficult types seen by the otolaryngologist. They may be summarized as follows:

1. Space abscess in the tissues of the face may occur concurrently or weeks following pericoronal or apical infection of third molar teeth.

2. Trismus is important in diagnosing space abscess, and odontopathic factors must be considered.

3. Abscess in regions limited by bone and muscle cannot produce typical swelling, and so diagnosis may be difficult.

4. Prudent surgical and supportive measures based upon understanding of the anatomy is preferable.

Oro-antral Fistula — A few clinical considerations of oro-antral fistula may now be mentioned. Many small fistula close spontaneously after extraction. The large rooted first permanent molar and the double rooted first bicuspid are favorites in fistula formation. Removal of upper third molars may also cause a fistula. A tiny opening into the sinus in this region is easily missed. This is seen only as a dimpling which simulates healed and scarred mucosa. The course of the tract may be tortuous and seen only with radio-opaque injection. Bizarre symptoms of sinusitis may occur.

Fistula may be associated with loss of a tooth or root into the sinus. Sublabial antrotomy may become necessary to retrieve it. As large as a tooth or root is, it may be hidden beneath the sinus mucosa and found only after frustrating search. Before exploration the lateral as well as the antero-posterior X-ray should be examined. The tooth may have been forced into the pterygomaxillary fossa instead of into the sinus.

Treatment of an oro-antral fistula may be accomplished by a pedicle flap from the buccal or palatal mucosa. Closure is dependent upon good drainage and absence of pathology in the maxillary sinus, and a nasal antrotomy may be performed where indicated. Simple approximation after freshening the edges of a fistula in a delayed closure is usually doomed to failure.

Facial Trauma — Facial trauma is a big subject. Only a very few odontologic matters of interest to the otolaryngologist can be mentioned here. Early tracheotomy is a preliminary consideration in extensive facial damage. In jaw fractures the simple examination of the mouth reveals much and may tell as much as the battery of X-rays usually ordered. Early treatment of certain jaw fractures, the same day if possible, is advantageous. Treatment may necessarily be delayed for days before the patient is able to tolerate therapy. Neurologic complications usually take precedence over definitive therapy. It is not necessary, however, to neglect the patient suspected of intracranial damage. Co-operation is needed in maintaining an adequate airway and in preventing aspiration of teeth or other foreign bodies. Sometimes temporary immobilization of fragmented bones is necessary. Middle face fractures may be reduced early. A shock state is overcome, and bleeding from the face often subsides when the bones are reduced.

Function of the jaws is based upon occlusion of the teeth, or proper interdigitation. It is to the advantage of the otolaryngologist to appreciate the importance of occlusion.

Retention of a tooth in the line of fracture is desirable when it may reasonably be saved. Conversion of an otherwise simple reduction into a more complex one can occur by the untimely removal of a tooth. In mandibular fractures, for instance, the only deterrent to extensive displacement of the vertical ramus of the mandible may be a lone molar at or near the line of fracture.

Blood in the ear in condylar fracture is not uncommon. Cranial damage must be suspected and due care taken. On the other hand, the blood may be from a tearing and fracture of the antero-inferior tissues of the ear canal by the condylar head as it is driven against the external canal. The otolaryngologist has a definite advantage in evaluation. Correctly used, the knowledge leads to a more prompt reduction of the jaw and anterior ear canal wall. Attention to the complication may avoid a later stenosis of the ear canal.

Many approaches to the treatment of jaw fractures have been proposed. Immobilization is essential. Intermaxillary wiring is useful and simple. Elastic traction, using small rubber bands between the upper and lower arches is an excellent way to reduce fractures.

Both open and closed reductions of the mandibular condyles and other parts of the jaws should be used as indicated. In extensive fractures other devices, sometimes quite complicated, may have to be used. No one way to reduce fractures is best, but many are equally good.

Aspirated Foreign Bodies — Dental foreign bodies may occasionally be aspirated into the air or food passages. Holinger in one series reported 2.5 per cent of foreign bodies in the respiratory system were dental objects. This is a surprisingly low figure, considering how often the oral cavity is invaded for dental treatment.

Aspirated teeth, or parts of teeth, account for the largest number of dental foreign bodies. Extraction under general anesthesia is a greater risk than extraction with local anesthesia. Roentgenographic evidence is helpful in locating dental objects. Not all foreign bodies are radio-opaque. Aspirated dental calculus, for instance, may be laden with infective material, and will hardly show up in a chest film. Silicate cements, used in the restoration of dental caries in anterior teeth are easily penetrated by Roentgen rays. Small pieces of plastic appliances, known as acrylic, will not produce a significant shadow. Metals and teeth will ordinarily be seen on neck and chest films, though they may be hidden by heart, liver or bone.

)steomyelitis — Osteomyelitis of the jaws is difficult to at, and the otolaryngologist will be called upon for help and insel by his medical and dental colleagues. The cause is ially of odontogenic or traumatic origin, and the mandible the most common site affected. In infants the maxilla is re commonly affected. During the acute stage much reliance placed on antibiotics. Emphasis should be given to the gical aspects after the acute phase has subsided. Sequesctomy and saucerization are useful and may be curative in treatment of osteomyelitis of the jaws. With the safeirds of pre- and postoperative antibiotics, the source of ection in bone can be more readily controlled.

adiation to the jaws may produce an osteomyelitis. rious, and sometimes sound teeth, may have to be removed ore the jaw is irradiated in order to avoid difficulty later Irradiated bone does not heal well, and very resistant and nful osteomyelitis results when teeth become infected and e to be removed.

ODONTOGENIC TUMORS.

Ve may now deal briefly with swelling due to cysts and iign tumors of odontogenic origin. Malignant tumors and se arising from non-odontogenic origin are not included.

)dontogenic Cysts — Odontogenic ectodermal cysts may se from the follicle of the tooth or from the periodontal mbrane. Several types of follicular cysts occur. The first he primordial cyst, which forms from the degeneration of stellate reticulum in the enamel organ before any calcifiion takes place. Another type is the dentigerous cyst. This ginates because of degenerative changes in the reduced imel epithelium of the developing tooth. It is a closed epilial lined sac formed about the crown of an unerupted tooth. is most commonly associated with impacted teeth, such as third molar or canines. Multilocular cyst is a third kind ectodermal odontogenic cyst. This forms from adjacent th germs or proliferation of epithelium.

'he other main group of cysts are those arising from the iodontal membrane and are named according to their location. Those at the apex of the tooth are termed periapical, while those located laterally are called lateral peridontal cysts. Their epithelium is derived from remnants of the sheath of Hertwig. Frequently these cysts are preceded by chronic proliferative inflammation. If acute infection is present, then all the symptoms of an acute abscess are present in the clinical picture. Cysts are important because of their slowly expanding nature. Much bone destruction and loss of function of the jaws may occur unless they are recognized and treated adequately.

Benign Tumors—As with cysts, benign tumors may also arise from developmental dental structures or from closely related peri-dental tissue. Classification may be complex. Only the more common types will be mentioned.

Inflammatory hyperplasia may often be confused with tumors. These are sometimes termed epulis. There is a tendency to recur if the tissue and underlying cause is not removed. Hyperplasia of the gingiva occurs because of irritation of a flange of a denture, irritation by an overhanging filling, decayed teeth, or calculus beneath the gingiva. Hyperplasia of a non-inflammatory nature may also occur in response to stimulation by certain drugs. This is most commonly seen in dilantin therapy for epilepsy.

Fibromas develop in the gingival or interdental papilla. They also form from periosteum or periodontal tissue, and may be penduculated or sessile. A fibroma may be seen at the maxillary tuberosity, and may be difficult to distinguish from malignancy when trauma has produced ulceration. Some fibromas show osteogenic activity, and may be extremely firm.

Another benign tumor is the peripheral giant cell tumor, also termed an epulis. The classical type has a bluish red tinge, due to vascularity and deposit of hemosiderin in the tissue. It frequently forms between the teeth, causing a separation and sometimes invasion of the underlying bone. It occurs as both a sessile and pedunculated tumor. Rare cases have occurred where fibrosarcoma has developed. In pregnancy similar tumors are found. It is believed these latter occur because of hormonal stimulation during pregnancy. They usually recede after parturition.

Among the most common benign osteogenic tumors are the torus palatinus and mandibularis. The palatal tumor is in the midline, and easily recognized. The mandibular tumors occur on the lingual surfaces of the mandible near the bicuspid teeth. They are usually smooth, very hard, and have been present for a long time. Often the patient is unaware of the mass in his mouth, until it has been called to his attention by his dentist or physician. These are considered to be hypertrophic bone or osteomas, and do not have to be removed unless they interfere with placement of a denture or other dental appliance.

SUMMARY.

Pain of dental origin, with some surgical considerations of interest to the otolaryngologist are discussed. Certain odontogenic tumors are considered.

It may be emphasized that the discussion presented is merely an introduction to that portion of medical science which the otolaryngologist finds necessary to evaluate and correlate with his own problems.

1648 East Ave., Berwyn, Ill.

UNIVERSITY OF ILLINOIS, COLLEGE OF MEDICINE.

The next Laryngology and Bronchoesophagology Course to be given by the University of Illinois, College of Medicine, is scheduled for the period November 5 through November 17, 1956. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois, College of Medicine, 1853 W. Polk Street, Chicago 12, Ill.

STATISTICAL ANALYSIS OF THE STAPES MOBILIZATION PROCEDURE*.

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Cleveland, Ohio.

When I started to put down on paper a comparison of the stapes mobilization procedure with the fenestration operation it was done in order that I might honestly and intelligently inform my patients as to what they might expect. It soon became apparent that I would have to call on some of my colleagues for help, because my experience was quite limited. A qu [illegible] nnaire on this procedure was sent to 14 men whom I e [illegible] knew were doing the procedure or whom I thought n [illegible] doing it. At the outset, let me make mention why I [illegible] send a questionnaire to Dr. Sam Rosen. Everyone [illegible] at he was the one who revived this procedure and for [illegible] time in otological surgery really made it work. His ch [illegible] s and results have been very adequately publicized. [illegible] ery kind to me when I visited him year before last, and I [illegible] ncly thank him for stimulating my interest in this [illegible] however, I wanted to find out what other persons could do [illegible] ith the procedure.

One of the remarkable things about this survey is that all the 11 questionnaires were returned. Three men said that they had not done the procedure as yet; therefore, the following analysis represents the combined work of these men:

Richard J Bellucci, M.D.; Edward H. Campbell, M.D.; Eugene L. Derlacki, M.D.; E. P. Fowler, Jr., M.D.; Victor Goodhill, M.D.; Frederick Harbert, M.D.; Robert Henner, M.D.; Howard P. House, M.D.; Charles E. Kinney, M.D.; Clair M. Kos, M.D.; George Shambaugh, M.D.; Theodore E. Walsh, M.D.

To these men, I give my thanks for their cooperation in this endeavor.

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The first question had to do with the number of cases which each man had done. The total reported cases was 1117. The second question was "Irrespective of the type of case, what is the percentage of the whole total in which there was a successful *audiometric* improvement of hearing within the first three to four weeks?" No attempt was made to set up a uniform standard of "successful improvement". They were asked to use their own criteria, because each one of these men had been doing the fenestration operation for a relatively long time. The third question was the same as the second question, except that the post-operative time was over six months. In analyzing the answers to these two questions, it was felt that it would be more accurate to balance each man's results against the number of cases that he reported; therefore, his percentage answer was multiplied by the number of his cases. These two figures were then totaled and the total number of cases was divided into the total percentages. The final figures were:

Successful three to four weeks—53 per cent.

Successful more than six months—40 per cent.

When one tries to obtain further ideas from these figures, particularly the added comments to these two questions by some of the men, there are two points that stand out: first, is that although the time consumed in doing this procedure is considerably shorter than the average time for fenestration operation the meticulousness required in order to obtain better results is of far greater importance than with the fenestration. The second point is, that as one does more and more cases his percentage of successful results goes up. For instance, in House's series of cases he reports that in his first 25 per cent of cases he only had 35 per cent successful results; in his second 25 per cent of cases his successes were 50 per cent, and in his last 50 per cent of cases his successes were up to 60 per cent. The observations alone have been sufficient to encourage me to continue this work and try to improve upon my earlier results.

The next question that I wanted some help on had to do with the idea that was created in the minds of the public by statements in newspaper and magazine articles to the effect that if the mobilization procedure did not work one could always do the fenestration operation on that ear.

It has always been my contention that the second most important factor in obtaining a really good result from a fenestration procedure was the maintenance of a completely intact and normally functioning ear drum. Of course the number one factor is the creation of the fenestra itself; therefore, I wondered whether the elevation of half of the drum by a mobilization procedure would leave a normal functioning drum after the incus has been removed and the head of the malleus clipped, as is done in the fenestration procedure.

In this series of 1117 cases, there were 60 per cent unsucce[illegible] results after six months, or 670 cases that were poten[illegible]es for a fenestration. There were reported 40 cases [illegible]d a fenestration done on a previous unsuccessfully [illegible]ed ear. This is only 5.9 per cent of the potential cases; [illegible]r of these 40 cases 33 were successful, which is an averag[illegible] 82 per cent, quite an admirable figure. When one tries t[illegible] some conclusions concerning this question he runs [illegible]eral unknowns. Of the 40 fenestrated cases (5.9 per c[illegible]he results were very good; but what about the 630 other pot[illegible]ial cases (91.1 per cent)? It is safe to assume that all of the[illegible] wanted better hearing or they would not have subjected [illegible]hemselves to the original mobilization procedure. Some of them have not had sufficient time interval after the mobilization Some of them have not had a fenestration because of economic reasons; however, all the men felt that, except in the very rare case, the condition of the ear drum after a mobilization attempt would not be a contraindication to a future fenestration. The 82 per cent of successful fenestrations in those cases that had been previously operated would tend to support this view.

The next question had to do with the amount of the bony posterior canal wall that had to be removed in order to have adequate vision of the stapes. Here again it was felt that each surgeon's answers should be balanced against the number of cases he had done. The results of this calculation reveal the following:

15 per cent required no bone to be removed from the posterior wall;

79 per cent required 1 to 2 millimeters to be removed;

6 per cent required more than 2 millimeters to be removed.

The next question had to do with the necessity of operating table audiometry. Nine of the 12 men whose cases are reported in this series felt that some sort of operating table hearing testing was necessary. Five of these nine felt that it was *absolutely necessary* to use some method of electrical audiometry at the time of the operation rather than depend on his own voice testing. It is interesting to note here that these five men reported higher percentage of good results than the other seven men.

Goodhill uses a standard commercial pure tone audiometer to do his operating table threshold testing. I think, but am not completely sure, that he uses only the frequencies of 500 and 1000. He simply puts a sterile towel over the auricle each time he tests the patient's response.

House has devised his own "Otometer," which is a pure tone instrument more adaptable to use in the operating room than a commercial audiometer. House feels that pure tone testing on the operating table is more reliable than electrically controlled word testing. He also feels that the amount of operating room improvement does not indicate the amount of improvement in hearing that the patient may have a month later. This idea is not shared by the other men.

Both Goodhill and House point out that in addition to an office pre-operative threshold for this particular ear, a pre-mobilization threshold must be established after the lower posterior part of the ear drum has been elevated and then returned to its normal position. This must be done each time one tests the hearing. The other three men who said operating table electrical audiometry was an absolute essential did not delineate the exact kind of testing that they employ; therefore, it is apparent that one must do the stapes mobilization under local anesthesia, and that one's vision and sense of touch are not sufficient to evaluate the degree of mobilization that has been accomplished.

My next question had to do with the most effective point to apply one's force in order to mobilize the footplate of the stapes. It is interesting to note that all 12 men seemed to be in agreement that fracture of either one or both of the stapedial crurae before the footplate is at least partially mobilized is still the biggest stumbling block to the success of this operation. Five of the 12 men reported that they used their first mobilizing force on the head of the stapes. Two men exert their initial force on the incus.

Goodhill advocates using a type of cement between the incus and the stapedial head if this joint should be dislocated in the original probing before one's actual mobilizing force is exerted. The other five men use their original force on the neck of the stapes as originally advocated by Rosen. There was absolutely no agreement as to the direction in which this force should be initially applied, that is, whether it should be a push or a pull; however, there was complete agreement that it must be in the axis of the two crura and that the exact direction of this axis must be ascertained before the original force is applied. This is a dictum that was first pointed out by Rosen. What one does after there is either visual or palpable evidence that there has been a fracture of one or both crurae, and before a hearing test shows that there has been some mobilization of the footplate, remains a big question.

Kos and House report that they use a small pneumatic hammer to produce a series of blows to parts of the rim of the footplate in case there has been a fracture of a crura. Three of the other men in this series report that they are working with this technique. It is obvious that this is the ultimate in technical surgical perfection, although House reports that by following this method he is able to mobilize at least part of the footplate in 95 per cent of his cases at the present time. There is one positive conclusion that can be drawn from consideration of this item: namely, this operation is *not the ten minute office procedure*, which idea has been bantered about too much in both medical and public discussion. There was rather universal agreement that no one should attempt this operation if they were not already fairly adept at doing the fenestration.

My next questions were directed towards the matter of complications. There were reported 19 cases of temporary acute otitis media. This is somewhat under 2 per cent of the total, and is certainly a tribute to the technique employed. There were no reported cases of semi-permanent or chronic otitis media and no cases of facial paresis or facial paralysis. There were three cases of progressive perception loss of hearing after this procedure which was assumed to have been caused by a hemorrhage into the perilymph. This is an insignificant number, but it can happen.

At this point in my questionnaire, I interposed a purely philosophical question: "Do you think that this operation will subsequently replace the fenestration operation?" Twelve answered "no" to this question, most of them emphasized in one manner or other; however, five of the 12 men said that they recommended that mobilization be tried first in all cases suitable for fenestration. There was rather complete agreement that mobilization had priority over fenestration in certain instances, such as economic reasons, cases that had a fair amount of perception loss but still a fairly good cochlear reserve, elderly patients or those with other constitutional diseases, and in cases that have had a fenestration in the other ear.

SUMMARY.

1. The complete cooperation and frankness of the surgeons that answered this questionnaire is something unique in medical annals.

2. 1117 cases of stapes mobilization are reported.

3. The immediate postoperative successful results were 53 per cent and the long range successful results were 40 per cent.

4. There is good reason to believe that some additional refinements in the technique of the actual mobilization will be developed which will certainly increase the chances of any one person obtaining a successful result.

5. Operating table hearing *testing is a must, and this should* be done audiometrically.

6. While the technique required to do this operation is more delicate than that required to do a fenestration, the time consumed is shorter, and *certainly the post-operative care of the* patient is reduced considerably.

10515 Carnegie Avenue.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

Washington, D. C., May 5-10, 1957.

The Officers and the Organizing Committee of the Sixth International Congress of Otolaryngology cordially invite you to attend the Congress which will be held in Washington, D. C., U. S. A., May 5 through 10, 1957.

REGISTRATION AND OPENING.

Any qualified physician may become a Member of the Congress by paying a registration fee of $25.00, U. S. A. Nonmedical personnel may register as Associate Members for a fee of $10.00, U. S. A. The registration fee includes the privilege of attending all official meetings of the Congress except the banquet for which an additional charge will be made. Those planning to attend the Congress should complete *Form A* and return it to the General Secretary.

IDENTIFICATION OF HEARING LOSS BY THE CLASSROOM TEACHER.

FRANK KODMAN, JR., Ph.D.,

Lexington, Ky.

The purpose of this investigation is to compare audiometric findings with teacher identifications of hearing loss to determine the accuracy of the latter method. Estimates of the incidence of mild-to-severe hearing losses in elementary school children based on studies by Curry,[1] Goldman[3] and Newhart[5] would be on the order of 10 to 25 per cent. Arbitrarily, mild losses may constitute audiometric thresholds 15 to 25 db at one or more frequencies in either ear. One would expect most of these losses to be conductive and worthy of medical attention from the standpoint of health and hearing conservation.

The teachers were asked to identify hearing losses 15 db or greater for the reasons cited above. Admittedly, the identification situation is unfair to both the teachers and the pupils; nevertheless, the classroom teacher is often expected to detect pupils whose auditory sensitivity is suspect. In a similar study, Curry[1] found that approximately one of four individuals referred for examination were found to have a loss of 30 db or greater at one or more frequencies in either ear.

PROCEDURE.

The general plan was to compare the percentage of teacher identifications of hearing loss with a valid criterion; namely, audiometric thresholds. Prior to the testing, each teacher was instructed to evaluate his pupils as having normal hearing or a mild-to-severe hearing loss. Each pupil was then given an individual audiometric screening test at 10 db at the octave frequencies 125-8,000 cycles and referred for pure tone threshold measurements if he failed to respond at one or more frequencies in either ear. The specific details of this procedure are to be found in an unpublished thesis by Goldman.[3] Com-

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parison data were collected on 665 elementary school children from a total sample of 709 children.

RESULTS.

On the basis of the audiometric findings, the pupils were classified into two groups: those with normal hearing, and those with losses 15 db or greater. The teachers selected 53 out of the 665 for audiometric evaluation. In other words, they felt that these had hearing losses. Of the total number tested, 540 had normal thresholds. The teachers erroneously classified 33 of these as hearing loss cases. Of the 125 comprising the hearing loss group, 20 were correctly identified by the teachers. This would mean that the teachers identified approximately one out of six hearing loss cases.

		AUDIOMETRIC TEST	
		% HEARING LOSS CASES	% SHOWING NO LOSS
TEACHER IDENTIFICATION	% HEARING LOSS CASES	a 3	b 5
	% SHOWING NO LOSS	c 16	d 76

Fig. 1 Tetrachoric table.

For purposes of discussion, the data from the two methods are shown below in a tetrachoric table in a manner suggested by Newby[4] (see Fig. 1).

Quadrant a shows the percentage of agreement between the two methods in relation to the correct identification of the hearing loss cases. Quadrant *d* shows the percentage of agreement as to the normal hearing cases. Quadrant *b* shows the percentage of cases with normal hearing who were referred by the teachers for examination. Quadrant *c* shows the percentage of hearing loss cases not identified by the teachers. To summarize, three per cent of the hearing loss cases were correctly identified by both methods. Five per cent were referred for examination but were found to have normal hearing. Sixteen per cent of the hearing loss cases were missed by the teachers. Seventy-six per cent were correctly identified by both methods as having no loss. For all practical purposes, the values in cells *a*, *b* and *c* show that teacher referrals leave much to be desired as a method of identifying hearing loss cases.

The next question raised is whether or not the teachers did better than chance. The teachers labeled 53 pupils as hearing loss cases. Of this group 33 were found to have normal hearing. Audiometric examination showed a total of 540 normals and 125 with a hearing loss. Chi square was computed and found to be 13.53, which is significant beyond the .01 level for one degree of freedom. Thus we can reject the null hypothesis of the differences being attributable to chance.

Examination of the audiometric results showed that the mean loss of those in the hearing loss group not identified by the teachers exceeded the mean loss in the identified hearing loss group by 15 db; furthermore, the six most severe hearing loss subjects were not identified by the teachers.

DISCUSSION.

The results of this study tend to support the findings of Curry, who reported that approximately one out of four hearing loss cases were correctly identified by the classroom teachers. In the present study approximately one out of six were correctly identified. The difference may stem from the criterion of hearing loss used in the present study. Since the teachers failed to detect five out of six of the hearing loss cases, it is interesting to speculate on some of the factors which may be affecting the judgments. Pupils exhibiting poor learning ability, inattention, asocial behavior, poor visual acuity, speech defects or frequent colds may be labeled as hearing loss cases. In a few instances, speech reception would be impaired sufficiently to furnish a reliable cue for identifying the hearing loss pupil. It would seem that the teachers, although performing better than chance, were basing the majority of their judgments on factors other than hearing loss. Hearing losses of the conductive type should produce variable sensitivity as a function of time. This factor would serve to alter the composition of the hearing loss group so that a given pupil may have a loss at one time and be within normal limits at a later period, or vice versa.

It should be interesting to compare parent identification of hearing loss with those of teacher identifications. This needs further study. One might hypothesize that the parents would outscore the teachers as detectors of hearing loss.

SUMMARY.

Teacher identification of mild-to-severe hearing losses was found to be better than chance but, nevertheless, inefficient when compared to audiometric findings. In addition to detecting a low percentage of hearing loss cases this method fails adequately to recognize normal hearing. Based on these findings and those of Curry, it is recommended that audiometric examination and not teacher identification be used to detect hearing losses in school children.

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University of Kentucky.

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University of Kentucky.

ABSTRACT OF THE PROCEEDINGS OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION

(Scientific Sessions)

May 13-14, 1956

SEIGNIORY CLUB, QUEBEC, CANADA

DR. BERNARD J. MCMAHON, President.

SAMUEL SALINGER, M.D., Abstract Editor.

HUMIDITY, A PROBLEM IN AIR CONDITIONING.

ARTHUR W. PROETZ, M.D.,
St. Louis, Mo.

To provide adequate relative humidity in the dwelling or office, to satisfy the needs of the nasal mucosa, is a problem that is not easily solved. With the optimum being between 40 and 45 per cent, one finds that central heating usually depresses the humidity to a point at which the mucous blanket becomes scanty and viscid, and ciliary action is slowed down. Figures assembled by Proetz from 19 cities during the month of January reveal an average outdoor humidity of 70 per cent and indoor humidity of 23 per cent. Even though the humidity can be raised to the necessary level within 30 minutes to two-and-a-half hours, depending on the apparatus employed, it would soon fall rapidly, due to losses through open doors, window cracks and even the walls themselves; consequently, the humidifying agent has to be kept going. Pans of water on the table or radiator are worthless. Commercial steam or cold vaporizers are somewhat better but not so effective as a teakettle on a gas flame.

As to humidity indicators, the sling psychrometer has been found to be the most reliable.

Central furnace type humidifiers have certain drawbacks arising from the dust which is scattered throughout the house. Unless means can be found for filtering the water this type of vaporizer leaves something to be desired.

DISCUSSION.

Dr. Lawrence R. Boies commented on the amount of valuable data assembled by Dr. Proetz on a very important subject. Although a practical solution of the problem of humidity control is still unavailable, it is comforting to know that the effort will continue to be made. Raising the humidity for therapeutic purposes has been satisfactorily accomplished in the management of acute laryngotracheitis in children. Dr. Boies has also found it practical to employ a humidifier in the postoperative care of patients whose nasal passages have been packed for a period following surgery; thus it is shown that humidity can be controlled in restricted areas in the care of the sick. If this could be extended satisfactorily to the home and the environment where people work, much would be accomplished in the prevention of the common cold and respiratory affections.

Dr. Edwin N. Broyles called attention to the plight of laryngectomy patients who suffer from dried mucus and crusting in the trachea during the winter months only. This proves the point brought out by Dr. Proetz as to the necessity of maintaining adequate humidity in the environment.

Dr. Thomas C. Galloway mentioned that low humidity in the Rocky Mountain area is well tolerated, whereas in the Middle West the same degree of humidity in the home is unhealthy. The explanation for this phenomenon may be in the dust factor which is present in the dry house. He inquired as to whether Dr. Proetz had an opinion on this point.

Dr. Proetz, in closing, offered as his explanation of the point brought up by Dr. Galloway that the distribution of pathogenic bacteria in the higher altitudes is much lower than lower down, and cited Grenfel's expeditions into Labrador, where there were no colds among the natives during the season when no ships arrived. Promptly, however, after these newcomers were landed, a severe epidemic of colds broke out among the natives with a number of deaths due, in all probability, to their inability to handle the bacterial invaders.

Dr. Proetz mentioned a very simple indication of too low humidity in a room, namely, the drawing of sparks when contacting objects.

HISTAMINE-RELEASING DRUGS.

F. C. McINTOSH, M.D. (By Invitation).

The human body contains approximately 300 mgm. of histamine concentrated mostly in mast cells along with heparin. Its action is threefold [illegible] cont[illegible]on of smooth muscle, increased gland a[illegible] permeability. A wide variety of drugs is listed, which are to greater or less degree histamine liberators. These include quaternary salts, morphine and other alkaloids, chemotherapeutic agents, circulatory drugs, sympathomimetic drugs, protein hydrolysates, basic amino acids and others. They seem to act through their affinity for the heparin stored within the mast cells. When a sufficient concentration has been reached the histamine-containing granules in the cells are lysed.

The relationship between the allergic or anaphylactic release of histamine and the release of histamine by drugs is a matter that has as yet not been satisfactorily explained. The author speculates that in the allergic reactions the antigen-antibody reaction may trigger some enzymic reaction leading to the formation of basic or polypeptide histamine liberators. "It is conceivable that by the local application of a histamine liberator a sensitized tissue could be depleted of its histamine stores for a long period, for instance the season of a particular pollen; the antigen-antibody reaction to the allergen could then still take place but would have fewer unpleasant consequences. The sudden unloading of the histamine depots would produce a severe reaction, but this could presumably be controlled with antihistamines and adrenalin."

The investigations conducted by the author and others are doing much to explain untoward reactions to certain drugs, and point a way to avoid or overcome them.

DISCUSSION.

Dr. Aubrey G. Rawlins reviewed the facts about histamine, its locale within the body and its physiologic action. One striking characteristic of histamine is its potentiality as a powerful endogenous agent capable of producing drastic physiologic and pathologic effects, yet under ordinary circumstances devoid of physiologic significance. Dr. Rawlins commented on the versatility of the mast cell and the various functions attributed to it by physiologists, such as its response to stress, its role in the production of ground substance of the connective tissue, its heparin-ilke polysaccharide which may act as an inhibitor of hyaluronidase, and now the storage and release of histamine as described by the essayist.

He submitted the question as to whether antihistamine drugs act as anti-hyaluronidase factors and whether hyaluronidase as well as histamine is liberated in cell damage, and plays an active part in inflammation; also, will hyaluronidase liberate histamine?

Dr. Sam E. Roberts inquired as to whether there is any synergistic action between heparin and histamine when given concurrently by intravenous infusion or are they antagonistic?

Dr. McIntosh replied to Dr. Rawlins' question to the effect that he knew of no evidence either for or against the activation of hyaluronidase in mast cell disintegration. In general mast cells do not survive very long in any real inflammation, although in mild chronic injuries they tend to increase in numbers. Histamine release is not of any importance in the maintenance of inflammation, because this release occurs explosively if at all, and it takes a long time for the histamine to be put back in the tissues. The duration of action of released histamine is so extremely short that the whole thing is over and done with in hours at the most, and cannot happen again for a long time.

It is possible that hyaluronidase is activated as a result of mast cell disintegration and is a subject worthy of further investigation.

As to Dr. Roberts' question, there is no appreciable synergism or antagonism between histamine and heparin; however, the histamine liberators form an insoluble complex with heparin, and heparin is a moderately active antagonist to the histamine liberators. It would be desirable if one could find a heparin analogue that might be more effective in this respect and still not have too much effect on blood clotting. Thus far such an ideal compound has not been evolved.

OTOLARYNGOLOGIC ASPECTS OF HYPOMETABOLISM.

JEROME A. HILGER, M.D.,

St. Paul, Minn.

Hypothyroidism in various degrees may be the basic etiologic factor in a wide variety of otolaryngologic conditions. The following are a few in which thyroid therapy has proven l as demonstrated in typical cases cited: Chronic ɾd edema, post-thyroidectomy paresis and edema, ıasal obstruction with discharge,, tubal catarrh, reerous otitis media, mucoproteinous edema of pharynues, vascular head pains, and endolymphatic dis- The diagnosis is reached through a careful history ıer laboratory investigation. Since the BMR test is ys reliable, it has been found that the radioactive ıtake is more dependable. Values of 5 per cent and ·e definite indications of hypothyroidism. Where the most accurate test is the determination of the ·otein-bound iodine.

ısis is laid on the importance of bearing in mind the y of thyroid deficiency in many of our otolaryngologic s where multiplicity of symptoms and chronicity tance to routine therapeutic procedures are dominant

DISCUSSION.

ncis W. Davison supported Dr. Hilger's thesis by citing three cases. One patient demonstrated the fact that hypothyroid ın be quite thin. In this case, hoarseness, nasal obstruction ıtion deafness were all due to low metabolism. The PBI was as against a normal of 5.3. Two grains of thyroid extract ght relief in about three weeks. In another case hearing due to middle ear effusion, yielded to thyroid therapy despite ıat the BMR was only —6.

patient who was obese also had recurring hearing loss due ear effusion. This patient was followed for 12 years and ffered a recurrence of the ear complaint when she discon- thyroid.

ison also noted that subthyroid patients usually like a room ɔerheated.

is F. Morrison called attention to the semantic aspect of this ıce there is a difference between hypothyroidism and hypo- ı. It is erroneous to assume that normal limits are between +10. This was proven in an experience with patients who italized for five days with BMR of —4 or —3, but who after ys showed —23 to —35. Dr. Morrison is opposed to the em- ꞉ription of supplemental thyroid therapy and warns against ce without suitable laboratory guidance.

꞉d Dr. Hilger why the serum cholesterol determination was ıned.

ıes M. Robb suggested that thyroid therapy be under the ı of the otolaryngologist rather than the internist, since the in a better position to evaluate the status of the auditory as well as the nasal passages and the larynx.

Dr. Hilger, in closing the discussion, pointed out that these patients are not putty-faced individuals as a rule. The PBI determination is a satisfactory guide to therapy, and should be followed because there is no dogmatic rule governing the optimum dose for each individual.

He agreed with Dr. Morrison that the BMR is inadequate and misleading, and frequently poorly done. If facilities are not available for a PBI determination it is a simple matter to send a specimen of the patient's blood plasma to any number of well-equipped laboratories and obtain a reading in a matter of a few days.

The matter of serum cholesterol determination was purposely omitted because it is only additive information, and the day was too nice to be spent in superfluities! It is merely a confirmatory test to the BMR and if reported normal leaves one unconvinced.

Dr. Hilger appreciated Dr. Robb's suggestion that the paper be read. In this instance one could come abreast in a short time of what took him much longer to assemble. The endocrine system is amazingly important in our field, much more so than in internal medicine. Otolaryngologists have the opportunity of making the primary diagnosis more frequently than in any other branch of medicine.

The point brought up by Dr. Morrison relative to the distinction between hypothyroidism and hypometabolism is well taken. He deliberately chose the title of hypometabolism and attempted to show that there is a hypothyroid hypometabolism or a thyrotic hypometabolism, but also a hypopituitary and a hypothalamic hypometabolism, as well as the hypothyroid type. Hypometabolism may be related to hypothyroidism, but all hypometabolism is not due primarily to hypothyroidism.

FUNCTIONAL DISEASES OF THE NOSE.

STEWART NASH, M.D.,

Rochester, N. Y.

UNUSUAL VIRUS DISEASES IN OTOLARYNGOLOGY.

P. E. IRELAND, M.D.,
and
T. J. MOLONEY, M.D.,

Toronto, Can.

Virus disease seems to be on the increase and presents a wide variety of manifestations, a number of which are classified by the authors. More in detail and relating particularly to the field of otolaryngology are APC viruses, cat scratch fever and atypical pneumonias. The adenoidal-pharyngeal-conjunctival virus was found to be mildly epidemic. In a Toronto epidemic apparently originating and being spread from swimming pools, a virus was recovered in several cases which was grown in tissue cultures of monkey-kidney epithelium or "Hela" cells.

Several cases of cat scratch fever were described, and emphasis was laid on differentiating the condition from mononucleosis, tuberculosis, Hodgkins' disease, tularemia, rheumatic fever, etc. The cat scratch skin test, which is done by injecting .1 cc of an antigen of known potency intradermally and noting the reaction after 48 hours, a positive reaction being the appearance of at least 5 mm. in diameter with an erythema of at least 10 mm. in diameter. The clinical course of the disease is described and typical cases cited.

The authors found an increased incidence of atypical pneumonias in areas where poultry raising was a prominent industry. Pigeons and turkeys were very abundant in a certain five-mile radius where these virus pneumonias were prevalent. It is hoped that further studies may identify the virus in turkeys and prove its transmission to humans.

THE VIRUS THEORY OF NASAL POLYP ETIOLOGY AND ITS PRACTICAL APPLICATIONS.

Francis L. Weille, M.D.,
and
Robert S. Gohd, Ph. D.,

Boston, Mass.

In an attempt to prove that a virus is responsible for the development and recurrence of nasal polypi, Weille and Gohn conducted a number of experiments in which polypi fluid was injected into the allantoic fluid of the chick embryo. This was found to result in increased proteins in the fluid. Passage of this fluid through succesive chick embryos produced the same changes through five successive passages, although a diminution in the virulence was noted beyond that point. This would seem to support the thesis that the infective agent was transmissible; however, in order to fulfil the last of Koch's postulates, namely to reproduce the disease in susceptible animals, attempts were made to produce gross or microscopic tissue changes in the nasal mucosa of experimental animals, all of which failed; however, the presence of a specific antibody concentration in the serum of polyposis cases and its absence in controls might be considered legitimate and important evidence. Studies will continue along the lines of devising a serological test to prove its existence.

The theory of virus etiology in polyposis, if fully proven, could well explain many of the manifestations of polyp growth, recurrence and frequent disappearance following diathermy to the stumps suggesting destruction of the intracellular viral agent.

DISCUSSION.

Dr. Lewis F. Morrison found that the common ground for discussing these two papers lay in the term "virus." He noted that no mention was made of the possible role of a virus in the etiology of laryngeal papillomata. He could not quite agree with Dr. Ireland about our present knowledge of viruses, which even in the opinion of virologists is quite meager. The points brought out with reference to the role of a virus in upper respiratory affections, adenoidal, conjunctivo-adenoidal, and the probable etiologic connection with the swimming pool are well taken.

Concerning the intradermal antigen test, there is a possibility always that the virus may not be sufficiently attenuated. Possibly the essayist may have more to say on this point.

The matter of turkey droppings as a contaminent in an area with a high incidence of atypical pneumonia seems to be a very practical consideration.

As to Dr. Weille's theory, it sounds convincing but perhaps only because nobody is in a position to refute it intelligently. Dr. Morrison felt that he could not quite divorce himself from the conviction that the allergic factors are more important than the virus. Since polyps are found in the mucosa of other organs than the nose, it would appear difficult to apply Dr. Weille's theory widely enough to cover all situations. As to the disappearance of polyps following post-excision diathermy's being due to heat killing the virus, it must be remembered that any tissue insult may be a factor in remission.

Fundamentally, Dr. Weille's work is most commendable. It is regrettable, however, that he was not rewarded by the discovery of a specific virus that could be readily identified.

He asked Dr. Weille whether the allantoic fluid had been centrifuged and examined under the electron microscope for the possible indentification of a virus.

Dr. Morrison called attention to the analogy between laryngeal papilloma and rabbit papilloma, in which a large virus was identified which is sensitive to tetracyclaine and terramycin.

Dr. Ireland replied to Dr. Morrison's query bearing on the potency of the antigen used in the skin test. The antigens are made from material taken from the so-called postule and, since the virulence is variable, it is better to take material from several lesions. As for turkey disease, he admitted that efforts at control were disappointing because of lack of co-operation on the part of the large packing companies in control.

Dr. Ireland paid tribute to his associate in the presentation, Dr. Terence Maloney, who did a great deal of the work in its presentation.

Dr. Weille stated that in the staff meetings at the Massachusetts Eye and Ear Infirmary, the privilege of becoming as violent as one pleased was accorded to all. It can, therefore, work both ways. Concerning Dr. Morrison's comment relative to the cause and effect of diathermy in the polyp cases, he failed to offer any alternative explanation to the one submitted by himself.

As regards allergy in relation to the etiology of polyps, rather than a virus, there are many people who do not believe that allergy has anything to do with polyps. It is true that the majority do believe in this relationship, but is it not possible that the allergy creates the basic immunologic response to the virus in the same way as hemolytic streptococci? Vaccines are still in vogue at certain centers, and there are those who are convinced that the immunologic responses to viruses are similar to those of the ordinary bacteria.

As to the use of the electron microscope, Dr. Weille's virologist has one available whenever he feels that it is necessary; however, up to the present he has not deemed it essential.

Mention was made of skin tests. These were tried, but without results. Since it is assumed that the antibody is the reagent resident in the skin mass, it is possible that a more thorough investigation along the line of the alphibetical APC. notation by Hubner and other workers might evolve a dependable serologic test; also if a few volunteers could be found it might be possible to produce polypi in the nose experimentally.

MANAGEMENT OF NON-MALIGNANT GROWTHS IN THE MAXILLARY SINUS.

O. E. Van Alyea, M.D.,

Chicago, Ill.

Polypi and cysts are the most common non-malignant growths found in the antrum. The former develop most often in the vicinity of the ostium as a result of irritation, which may be an allergy or a pathogen from infection. They may grow into the sinus cavity or out of it through the ostium, appearing in the middle meatus or even growing backward into the choana.

Cysts may be either the simple mucous type or may originate from the dental appendanges in the form of radicular or follicular cysts. Frequently they are symptomless but at times may be responsible for local numbness, nasal congestion and frontal headaches.

Diagnosis in all cases can readily be made through the use of contrast media with X-rays. Most cysts are found in the floor of the antrum or the lateral walls, and are usually smooth and almost spherical in outline. Polypi, on the other hand, are more irregular in outline and most common along the vaso-antral wall. If they cannot be controlled by antiallergic therapy they should be removed via a sublabial approach. The antrum mucosa is in most cases left undisturbed.

Cysts may at times be eradicated through an intranasal antrostomy, although in many instances it is necessary to resort to the Caldwell Luc operation because of their location and size.

DISCUSSION.

Dr. Robert Goodale emphasized the point that cysts of the antrum *are not to be ignored or minimized, particularly if the patient has* symptoms referable to the one side. He cited a case of uveitis which failed to respond to therapy until an apparently simple cyst of the antrum was removed, revealing pus and a predominantly pneumococcus infection. *Too much reliance should not be placed on the antibiotics* when infection is present along with a cyst.

Polypi and cysts are common findings. Dr. Goodale favors the removal of polypi developing within the antrum via the canine approach. It is not necessary to remove the mucosa entirely. The surgery should be limited to the presenting pathology. Additional drainage through an inferior meatus opening is not always necessary. He agrees with Dr. Van Alyea that the possibility of malignancy in all these cases should never be overlooked. *All material removed should be sent to the pathologist.*

Dr. Lewis F. Morrison inquired as to the presence of organisms in the cysts. *Previous reports on this item have been contradictory.*

Dr. Robert S. Priest *wanted to know whether Dr. Van Alyea feels* that surgical exploration is indicated in all cases of cysts. He has had the experience of passing up some of these benign growths only to have the patient return complaining of pain, and subsequent biopsy *revealed mixed tumor.*

Dr. Van Alyea replied to Dr. Morrison's query by stating that cultures have been done routinely, but that he had no significant report to offer; besides, in many cases the cyst was opened via the intranasal route, often merely as a result of antrum irrigation.

EXPERIMENTAL TREATMENT OF RECURRENT CARCINOMA OF THE NASOPHARYNX WITH ELECTRODESSICATION, RADIOACTIVE COBALT AND X-RAY RADIATION.

FRANCIS A. SOOY, M. D.,

San Francisco, Calif.

Six cases of recurring inoperable carcinoma of the nasopharynx previously unsuccessfully treated with irradiation were reported by Dr. Sooy. These were treated by electrodessication and local intranasal radiation with radioactive cobalt. The greater part of the septum was removed to afford more space for the transnasal manipulations. Three of the patients were alive and free from signs of recurrence more than six years following therapy. Subsequently ten additional cases were similarly treated, with five of them surviving for periods of from six months to four years.

Cases showing clinical or X-ray evidence of extension into the base of the skull were excluded.

The electrodessication was carried out with caution, lest excessive heat cause damage to the pituitary and optic nerves.

The radioactive cobalt was applied at 1 cm. distance from the area, via a Foley urethral catheter, the bag being distended with radio-opaque solution to permit accurate placement of the cobalt. The dosage administered was 4000 to 6000 r.

Although considerable dryness was observed, there was very little crusting after the initial slough and sequestra had been removed.

DISCUSSION.

Dr. Harry P. Schenck felt that this presentation teaches us not to be too pessimistic about some of these malignancies. He has always felt that electrocoagulation was not employed widely enough in otolaryngological practice. One reason may be the fact that local anesthesia often limits the extent and degree of the coagulation as compared to the freedom afforded under general anesthesia.

Concerning the freedom from crusting following removal of the septum reported by the author, Dr. Schenck feels that there must be some difference in the influence of climate, because the cases he has seen in Philadelphia manifest marked crusting for months.

The low incidence of metastases in the cases reported is rather striking in view of the size of the primary lesions In his cases, he has not been so fortunate.

The advantage of the technique described is obvious since it affords a wide approach to all the affected area.

In this connection, Dr. Schenck suggested that this procedure need not be restricted to carcinoma alone He had had a good result in an extensive chordoma with destruction of the sella turcica by wide electrocoagulation applied through both sides of the nose as well as behind the palate He did not destroy the septum.

Dr Thomas C. Galloway has for many years been enthusiastic about the use of electrocoagulation.

It is important to know something about the character of the tumor, many of them being lympho epitheliomas or transitional cell carcinomas, both very malignant with early involvement of the glands He noticed that the authors used X-ray therapy in some of these cases and wondered how much of the good result might be attributed to this medium rather than the cobalt. Removing the septum to afford an approach to the tumor impressed him like Lamb's story about the Chinaman who burned a house down when he wanted a roast pig.

He had found it practical to use Dr. Mosher's old incision, modified by a trapdoor through which the carcinoma involving the frontals, antrums and sphenoids could be completely visualized and destroyed; also at times it is just as feasible to approach the area via a transpalatal incision.

It was his impression that tumors arising from the septum were usually of the squamous cell type, being less malignant and more effectively destroyed by a less mutilating procedure.

The outlook for the more malignant types is not so dark as previously considered He has had the good fortune of seeing some of these cases cured in recent years and considers the combined use of electrosurgery and radiation as being responsible He assumes that the essayists were using the biterminal electrodes which penetrate more deeply than the superficial dessicating current.

Dr. Sooy replied that their dessicating unit is adjusted to achieve a slough of about 7 cm of bone and mucosa

The transpalatal approach was considered but rejected in favor of the one described, because they wanted to preserve a wide aperture for inspection of the site in the postoperative period.

In addition to the symptoms of nasal obstruction and bleeding and cervical metastases, they frequently found early indication of the growth in the ear symptoms such as unresolved serous otitis media. In three cases a prophylactic biopsy in Rosenmuller's fossa yielded positive evidence of lymphadenoma.

CILIARY STREAMING THROUGH THE LARYNX AND DISTRIBUTION OF LARYNGEAL EPITHELIUM.

A.C. HILDING, M.D.,

Duluth, Minn.

Studies were made on the distribution of squamous and ciliated epithelium in five human larynges removed at autopsy and in a number of recently killed calves. The results corresponded to the generally accepted pattern except that above the vocal cords the squamous epithelium is apt to occur in islands or peninsulas, surrounded by ciliated columnar epithelium. Further observations on the patterns of ciliary streaming seemed to indicate two streams coming up from the trachea. The posterior one passes between the arytenoids to the rim of the larynx. The anterior stream is split into two parts at the squamous cell barrier of the cords, flowing backward beneath the cord to the arytenoids.

The absence of ciliary currents in certain areas of the vocal cords seems to suggest a predisposing factor in the occurrence of carcinoma on the cords in 80 to 85 per cent of all malignancies of the larynx. Whether the inhaling of cigarette smoke or other noxious vapors in the atmosphere is as important etiologic factor as other factors such as vocal trauma, etc., is a matter that requires further study and analysis.

DISCUSSION.

Dr. G. Edward Tremble mentioned the statement found in most standard textbooks that the mucous membrane of the human larynx varies according to the functions performed; thus the difference between the squamous epithelium of the true cords and the ciliated columnar types with goblet cells in other areas. It is important to remember that the interarytenoid space contains both types of epithelium. When the arytenoids approximate, the tissue between them is thrown into folds, the ridges being covered with squamous epithelium and the valleys with columnar ciliated. Nature has provided a similar arrangement in the nasal mucosa. Dr. Tremble described the ciliary streams coming up the trachea, the posterior stream going directly to the interarytenoid space and the anterior passing below the cords to reach the same area. Convergence of the streams at this point probably explains the high incidence of tuberculosis lesions in the posterior portion of the larynx.

Dr. Tremble has used dicalcium phosphate powder which turns bright orange color when in contact with moisture and serves as a good guide in determining ciliary streaming. The powder thus applied in the trachea seems to follow a spiral rather than a straight course, which may be due to the movements of the trachea in respiration.

Dr. Arthur W. Proetz was impressed by the finding described by Dr. Hilding where the ciliary stream causes material to accumulate in the center just below the cords before spreading and passing posteriorly. This is just the area where subglottic lesions develop which are frequently overlooked, however, it must be noted that in the specimens observed by Dr Hilding, the cough phenomenon was absent, of course, so that one cannot precisely evaluate the results He would like to see this part of the investigation carried on with this reflex in mind.

The point brought out by Dr Hilding regarding the mucous anchors of thick mucus coming out of the glands, and their influence in retarding the movement of the mucous blanket, ties in with his own observations on the effects of altered viscosity on this function, often the result of inadequate humidity and at times the result of limited thyroid activity, as pointed out by Dr Hilger.

Dr. Proetz was wondering what happened to the spiral phenomenon described by Dr. Franklin, of London, and just mentioned by Dr. Tremble It calls to mind the beautiful demonstration of spiral streaming in the sinuses which Dr. Hilding demonstrated some time ago

Dr. Thomas C. Galloway inquired how fixed and persistent is the pattern of squamous and ciliated epithelium, and how much metaplasia occurs as a result of trauma, chemical irritation, etc.

Dr Hilding called attention to differing predilection in sites between tuberculosis, which occurs more frequently in the posterior portion of the larynx, whereas carcinoma was more common in the anterior. The facts are still not entirely clear.

Since these experiments were performed in dead animals, the findings would differ from those in the living because of the absence of the respiratory movements and changes in dimensions Spiral streaming was not observed in these animals; however, in one human trachea which he obtained some months ago there was a very definite spiral flow

Regarding the question brought up by Dr. Galloway, from data obtained in similar experiments in the nose, he could only infer that the pattern of epithelium changes according to the stresses, normal and pathologic, which occur or are induced in the areas under observation.

* * *

Dr. John A Murtagh introduced Dr. Campbell, guest speaker, with whom he had been associated for ten years in a study of the basic factors of the larynx and recurrent laryngeal nerve, and expressed his appreciation of his valuable contributions to the knowledge of the subject.

ELECTRICAL MANIFESTATIONS OF RECURRENT NERVE FUNCTION.

C. J. CAMPBELL, M.D.,
and
JOHN A. MURTAGH, M. D.,
Hanover, N. H.

A number of experiments were conducted on goats to determine the presence of mixed components in the recurrent laryngeal nerves. These nerves were dissected free in the *anesthetized animals. "Stimulating electrodes connected to* a Gross square stimulator and recording electrodes leading to a pre-amplifier, amplifier and cathode ray oscillator were placed on the nerve."

The nerve was crushed distally, and the recordings of repetitive stimulation, as traced in the oscilloscope, photographed on a drawing board for enlarged tracings.

Much of the information thus gleaned pointed to the existence of proprioceptive afferent nerve elements from the laryngeal musculature. These investigations would seem to support the contention of Sherrington that motor nerves possess afferent as well as efferent fibers. The reciprocal inhibition shown by antagonistic laryngeal muscles adds further weight to this thesis.

The recordings were made at the peripheral end of the nerve, thus eliminating any influence from the tracheal and esophageal branches.

The experiments also appeared to demonstrate a double nerve supply to the cricothyroid muscle (superior laryngeal and recurrent).

DISCUSSION.

Dr. Louis H. Clerf speculated on the question as to how far one can translate findings in experimental animals to the human being. A case in point is the finding by one investigator of motor fibers distributed to the arytenoid muscle from the superior laryngeal nerve in man, whereas other investigators, failing to find these fibers in the dog, assumed the same to be true in man. It is of interest that pressure applied briefly to the recurrent nerve does not interfere with its conductivity; however, he suspected that remote effects would arise as shown in several patients operated upon for pulsion diverticulum who developed unilateral paralysis apparently due to pressure of metallic retractors applied for 10 to 15 minutes.

Dr. Clerf was in agreement that one cannot rule out reflexes as a possible explanation of some of the results obtained in these and similar studies. They demonstrate that the degree of stimulation employed greatly influences the reactions, the interpretation of which is taken into account in the final analysis of the results.

While the question of proprioceptive elements in the muscles of the larynx has not been proven, having in mind the remarkable reciprocal relationship of the external ocular musculature and its similarity to the two halves of the larynx, it would appear that further study of the end organs in the laryngeal musculature is necessary.

Dr. Lewis F. Morrison stated that in view of the variable types and numbers of bifurcations of the nerve in the neck, it might be of value to do the laryngectomies from above downward rather than the reverse; also some knowledge might be gained by dusting some powder in the trachea and following its course while the procedure is being carried out.

[illegible] inquired of the essayists as to the formula for the amount of voltage employed and the duration of its application.

Dr. Campbell replied that if the facilities were adequate one might well repeat the experiment of Ogura and Aulenton in the superior laryngeal nerve stimulated *in situ* and following laryngectomy using the extirpated nerve as they have done in the goat.

He believed that it is necessary to have square wave stimulators, the Grace instrument being the type he employed. Stimulus applied for one-tenth of a millisecond at one to three volts should be sufficient for most of the fibers; however, if one is desirous of observing the individual muscles the whole gamut of strength and duration would have to be used to get anything worth while.

Reverting to Dr. Clerf's point about the pressure, in his experiments it was applied for only a few minutes. Others have used pressure for longer periods and obtained a blockade of the function of the larger fibers. Lexal devised an instrument which would block out the largest fibers but retained function of the slower ones in the Gamma group, but finally decided that just pinching the nerve between the thumb and finger for 30 to 60 minutes would accomplish the same thing. The time element is important, and it may be that significant data may have been missed by not carrying the experiment over a longer period of time.

Referring to another item mentioned by Dr. Clerf, he did attempt to examine the muscles to see if these were spindles present, which was not the case; however, they did take the precaution of examining the muscles, in which everyone agrees that they are present. So the difficulty must have been with them and their present techniques, which he hoped would soon be improved.

SUBMUCOSAL COMPARTMENTATION OF THE LARYNX.

JOEL J. PRESSMAN, M.D.,

Beverly Hills, Calif.

The subject of this presentation was suggested to the author in his studies of the fate of radioisotopes injected into the larynx of experimental animals. It resulted in the observation that the submucosal area was divided into a number of compartments. Fluid injected in these spaces failed to pass from one to the other. Dyes were injected into certain areas of the larynx during the course of a laryngectomy and the distribution studied at the conclusion of the operation. Numerous colored slides were shown which demonstrated that the dye failed to cross the midline from one compartment to its opposite fellow. This applied to the epiglottis, vocal cords, ventricular bands and arytenoids. The same results were obtained in similar experiments on the living dog. The direction of the spreading dye was studied in a variety of situations and demonstrated the areas and extent of the various compartments. These findings would seem to coincide with the results of Dr. Hilding's experiments on ciliary flow, and also are in line with the distribution and direction of the lymphatics. They also suggest an explanation of the method of spread of carcinomas arriving in various locations within the larynx.

DISCUSSION.

Dr. Paul H. Holinger brought up the point about the role of the small holes in the cartilage of the epiglottis as an avenue of escape for the fluid from one compartment to another, a matter that would have to be determined by the microscope rather than by gross appearance, in view of their tiny caliber.

In some of the slides shown it appeared to him that the fluid did cross the midline, and he wondered whether it was due to the lymphatic distribution or perhaps an intracellular pickup. He thought the compartments might be better delineated with fluid injected under a lesser degree of pressure. Considering the clinical interpretation of edema of the larynx, its favorite sites and manner and spread, this is all understandable on the basis of anatomy, the phonating margin of the cord being tightly bound down and the subglottic area being loosely areolar; also it is conceivable that the direction of the various muscles and their compartments may have much to do with the distribution of injected fluids.

It is of vital importance to recognize the role of the lymphatics in the spread of carcinoma in the larynx, which predispose certain areas to a carry-over from one side to the other.

Dr. Chevalier L. Jackson considered this presentation as another example of a new anatomical concept having considerable clinical significance He was impressed by its analogy to the subject of segmentation of the lung, in which he has been deeply interested for some time The clinical application of this newer knowledge of anatomy has had a direct bearing on the surgery of this organ; however, in the matter of clinical application of Dr Pressman's findings to the question of carcinoma of the larynx, it would be premature to apply them too closely at this time, although the possibilities in other directions are quite evident.

Dr. Pressman agreed with Dr. Jackson in the matter of cancer in relationship to his experiments; nevertheless, it is a subject to which some attention should be directed. Dr. Holinger's point about the spread of the dyes across the midline has merit, because we know that structures such as nerves and vessels do pass from one side to the other, thus affording possible channels for spread. It should be remembered, however, that the degree of pressure and the amount of fluid injected would have much to do with either the limitation within the compartment or its forcible spill-over into the opposite side

This study has at least demonstrated to him in his work with radioisotopes that a mass injection in one space will not accomplish the results obtained when several spaces are injected.

CANCER OF THE LARYNX.

FERNAND MONTREUIL, M.D., (By Invitation).

Analysis of a series of 70 cases of carcinoma of the larynx covering a period of three-and-a-half years was presented by Dr. Montreuil. The cases were divided into three groups: intrinsic (confined to the vocal cord); extrinsic (within the laryngeal box); and extra-laryngeal. It was pointed out that the symptoms were more dependent upon the site of the growth than its size and extent. For instance, vocal cord lesions produce hoarseness in the earliest stages when the lesion is small, whereas in larger lesions away from the cords, the disturbance of the voice may be delayed.

Direct laryngoscopy and biopsy are essential for diagnosis.

Because of the unpredictability of the virulence of various lesions, it is considered advisable to do a neck dissection in all cases of extrinsic and extra-laryngeal lesions.

The author's experience with laryngectomy after extensive irradiation was not good, the wounds breaking down early with delayed healing.

Laryngectomy is usually done under local anesthesia except when preceded by tracheotomy or when neck dissection is contemplated. The T-incision is preferred, with removal of the hyoid bone and strap muscles. Early ambulation is encouraged.

DISCUSSION.

Dr. Henry B. Orton prefers to classify his cases according to the anatomical site of its origin. A record of a series of cases which he presented at a recent meeting of the Academy showed that his best results following surgery were in the following order: Cords, epiglottis, ventricles, arytenoid, subglottic, epiglottis and tongue, ary-epiglottic fold and pyriform sinus.

Dr. Orton called attention to one area frequently overlooked, namely, the vallecula, where exposure may be difficult if the epiglottis is close to the tongue. In these cases a transhyoid pharyngotomy will often spare the patient a total laryngectomy. Dr. Orton prefers general anesthesia, because there is less shock to the patient when he realizes he can no longer talk. Also he prefers a "U" incision, which affords better exposure.

Dr. Montreuil's operative mortality of 4 per cent seems a little high in view of his own experience in 450 cases with a mortality of only 1.3 per cent.

As for radiotherapy, he is averse to its use either pre- or postoperative, because of possible complications such as radionecrosis.

Dr. Francis E. LeJeune was interested in one item touched upon by Dr. Montreuil, namely, the possibility of implanting the carcinoma lower down in the bronchiopulmonary tract by way of the diagnostic bronchoscope. In this connection it is worth while mentioning the respect with which general surgeons treat the primary lesion, taking care that the knife and frequently the gloves used for the incision are discarded in favor of fresh ones.

Dr. Edwin N. Broyles inquired as to why the author fed his patients by intravenous injection rather than via an esophageal tube.

Dr. Montreuil agreed with Dr. Orton that various classifications of laryngeal carcinomas are fequently puzzling. It has been his policy to describe the lesion according to its exact location.

Concerning his reluctance to use a feeding tube, he was probably influenced by the fact that it causes the patient considerable discomfort, both in the nose and in the pharynx. This was particularly true of the old rubber tubes; however, the newer polyethelene tubes are much less irritating and have been used by him in cases where tube feeding has to be carried on for some time because of a fistula.

BENIGN GROWTHS OF THE VOCAL CORD: CLINICAL vs. MICROSCOPIC DIAGNOSIS.

SAMUEL SALINGER, M.D.,
Chicago, Ill.

An analysis of over 250 consecutive biopsies from the vocal cords yielded some interesting data from the viewpoint of comparing the preoperative clinical diagnosis with the microscopic findings. It was shown that the tumors which were definitely neoplastic, such as carcinoma, papilloma, etc., were generally recognized by their gross appearance and were confirmed by characteristic microscopic pictures; however, in the case of non-neoplastic growths the divergence was remarkable. Dr. Salinger found fault with the diagnosis of "vocal cord polyp" because in 81 cases so diagnosed, not one was given such a designation by the pathologist. In a considerable number of this group the growth was labeled "edamatous fibroma," but the remainder covered a wide variety of conditions, which were named according to the predominant changes found in the various components of the tissue. The author is convinced that there is no characteristic histologic picture peculiar to the so-called polyp and that the term is, therefore, misleading, since it implies a definite entity, which it isn't.

DISCUSSION.

Dr. Francis E. LeJeune called attention to a description of polyp pathology in Coates, Schenck and Miller's recent "Otolaryngology," indicating a characteristic stroma of edematous tissue with prominent vascular changes. Similarly, Morrison and Eggston and Wolf found these growths to be characteristic of a traumatic or inflammatory nature with frequent thrombosis of blood vessels, which has given rise to the diagnosis often given by pathologists of "angiomatous polyp." Dr. LeJeune agreed that these are benign growths, but would prefer to retain the designation of "polyp" as a challenge to pinpoint our diagnosis.

The disparity between clinical and histological diagnosis in the 81 cases cited by Dr. Salinger is not entirely the fault of the pathologist, but should be attributed to the lack of unanimity of thought between the patholgist and the laryngologist.

Dr. James M. Robb recalled having read a paper in which he termed the controversial growth mentioned by Dr. Salinger a "hemorrhoid of the vocal cord." He was impelled to so designate it because of the report of the pathologist, who said "in my opinion frequently there is a dilated vessel, a dilated blood vessel, that in time undergoes changes; chronic irritation always produces fibrous tissue which always contracts." Dr. Robb would be very glad to find some other label to attach to this condition, but because it seemed to be mainly a varix he thought "hemorrhoid" was not too bad.

Dr. Salinger was glad to have the references mentioned by Dr. LeJeune, in which the authors seemed to agree on a typical microscopic picture; however, from his own experience he is not convinced. The fact that the pathologist in examining a polyp rarely calls it a "polyp" indicates the lack of uniformity in the histologic analysis. Until pathologists can agree on a characteristic histologic picture peculiar to the polyp, he is unwilling to employ that term, and would prefer naming it simply "a benign growth of the vocal cord."

PRIMARY MALIGNANT TUMORS OF THE UVULA.

Fred Z. Havens, M.D.,
and
Rich C. Ye, M.D.,
Rochester, Minn.

Primary malignancy of the uvula is very rare. Havens and Ye were able to collect only seven cases that were observed at the Mayo Clinic from 1919 to 1952. During the period there were some 1,500,000 patients admitted to the Clinic. In 42.8 per cent of the cases secondary lesions were present, which led to disaster. The lesion is usually an ulcer or a granulated area on the uvula which appears indurated and stiff rather than flabby, as is the normal uvula. The authors advise removing the organ by surgical diathermy. Neck resection was not carried out in cases where there was no evidence of lymph node involvement.

Only two of the cases survived, one for four years and the other for one year when last heard from.

DISCUSSION.

Dr. Leroy Schall, reviewing his records of 12 cases seen since 1935, finds himself in agreement with the essayists. He is impressed by the absence of early symptoms in some of these cases. In one instance it was discovered by the patient's dentist. Unfortunately, many of these patients come in too late, and it is difficult to determine whether the lesion was primary in the palate or in the uvula. Eight cases were treated by electrosurgical removal; six showed no recurrence for periods of one, two, six, seven, eight and ten years. Four cases were treated by irradiation. Three showed no recurrence after three, four and six years respectively.

Dr. James M. Robb inquired whether Dr. Havens removed the soft palate. He noted that one patient was dead, but was wondering whether the others have been followed up to date. Certainly a malignant lesion of the soft palate is a serious problem.

Dr. Havens said that, like Dr. Schall, he was unable in many cases, judging by the records, to determine just where the lesion started; therefore, he omitted these cases from his series. It was his desire to point out the danger of this lesion even when it was localized to the palate alone. Thus, future investigators could be guided in their evaluation of the lesion by the knowledge that this series included only those cases in which it was positively demonstrated that they were primarily in the uvula.

Too much emphasis cannot be placed on the importance of careful examination. One case cited had passed through the hands of a very competent otolaryngologist who was very much embarrassed at having missed the lesion. In this case it involved only the very tip of the uvula, but on palpation seemed to be firm enough to excite suspicion.

Replying to Dr. Robb's question, the answer would be to take off as much tissue as necessary, even if it meant removing the entire palate.

In the one case which was operated on the lesion involved only a third of the uvula. All the others were years back.

USE OF THE LARYNX IN REPAIR OF CICATRICIAL STENOSIS OF ESOPHAGEAL INTROITUS.

JOHN B. ERICH, M.D.

Dr. Erich reported a case in which the larynx was attached to the esophagus to bypass a structure at the introitus resulting from radiation necrosis, following therapy for a carcinoma. The patient was a man of 68. The lesion, a large malignant tumor, Grade III, occupied the larynx and hypopharynx, causing marked obstruction; also there was an enlarged cervical node.

After tracheotomy the growth was destroyed by surgical diathermy following which radon seeds were implanted at the site of operation as well as into the lymph node. Feeding was accomplished through a tube. Marked edema and perichondritis with sequestration ensued. Eighteen months later there was no evidence of recurrence.

It was found that the vocal cords were in complete abduction, which was favorable for the technique which followed. The larynx was split and severed from the trachea, which was dissected free from the esophagus for a distance of 3 cm., and its free margin then sewed to the skin. The esophagus was severed just below the stricture and attached to the mucosa of the larynx, which then served as its upper end. Since the cords were well abducted, soft food passed readily, and within a short time the patient was able to swallow any type of well-masticated food. Three years have elapsed, and the condition is satisfactory, with no indication of any recurrence.

MECHANICAL APPLIANCE FOR CICATRICIAL STENOSIS OF LARYNX AND UPPER PART OF TRACHEA: REPORT OF A CASE.

JOHN B. ERICH, M.D.

Stenosis of the larynx, due to perichondritis following an automobile accident in a man of 26, was treated by excising of the scar tissue through a thyrotomy and the implanting of a Thiersch graft over a sponge. After the graft had taken, an acrylic obturator was prepared and worn by the patient. At the time of operation it was noted that the esophageal wall protruded into the lumen of the larynx due to loss of a great portion of the cricoid cartilage. A small fistula developed which failed to heal, despite closure with a flap of mucous membrane. It was then necessary to cover the area with skin, which was implanted via a tube flap obtained from the clavicular area, and brought up into the larynx. The fistula was thus closed, but the redundancy of the skin flap so narrowed the lumen as to prevent adequate breathing. Consequently, an acrylic tube was fashioned with a ring-shaped extension protruding through the tracheostomy, through which the tracheal cannula could be inserted. A hole in the tip of the cannula permitted air to pass from the trachea into the upper air passages so that breathing through the nose and mouth could be accomplished when the external orifice of the cannula was corked. The cannula and acrylic device could be easily removed for cleansing and has been worn with satisfaction for two years up to date.

ANNOUNCEMENTS

The American Otorhinologic Society for Plastic Surgery takes pleasure in announcing the following:

2. A course in Plastic Surgery of the Head and Neck will be given in New Orleans, Louisiana, February 17-22, 1957, at the Louisiana State University. This course is to include cadaver, didactic and wet and dry clinics.

Early registration is recommended because of the limited facilities. Judging from our previous courses there will be oversubscription.

For further information address Dr. L. J. Feit, Secretary, 66 Park Ave., New York 16, N. Y.

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SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

The meeting dates of the Sixth International Congress of Otolaryngology are again emphasized as May 5th through May 10th, 1957. The scientific program for the Plenary Sessions is now complete and is as follows:

CHRONIC SUPPURATION OF THE TEMPORAL BONE.

OPENERS: Marcus Diamant, Central County Hospital, Halmstad, Sweden—Anatomical Etiological Factors in Chronic Middle Ear Discharge.

Luzius Rüedi, Zurich, Switzerland—Pathogenesis and Treatment of Cholesteatoma in Chronic Suppuration of the Temporal Bone.

Horst Wullstein, Director, Otolaryngological Clinic, University of Würzburg, Germany—Surgical Repair for Improvement of Hearing in Chronic Otitis Media.

DISCUSSERS: A. Tumarkin, Liverpool, England; Juan Manuel Tato, Buenos Aires, Argentina; T. E. Cawthorne, London, England; Fritz Zöllner, Freiburg, Germany.

COLLAGEN DISORDERS OF THE RESPIRATORY TRACT.

OPENERS: Hans Selye, Director, Institute of Experimental Medicine and Surgery, University of Montreal, Faculty of Medicine, Montreal, Canada.

Introduction:

Michele Arslan, Padua, Italy—The Upper Respiratory Tract.

Leslie Gay, Physician-in-Charge, Allergy Clinic, The Johns Hopkins Hospital, Baltimore, U. S. A.—The Lower Respiratory Tract.

DISCUSSERS: Victor E. Negus, London, England; Branimir Gusic, Zagreb, Yugoslavia; Aubrey G. Rawlins, San Francisco, U. S. A.; Henry L. Williams, Rochester, Minn., U. S. A.

PAPILLOMA OF THE LARYNX.

OPENERS: Jo Ono, Tokyo, Japan—Etiology.

Plinio de Mattos Barretto, Faculty of Medicine, University of Sao Paulo, Brazil.

Diagnosis:

F. C. W. Capps, London, England—Therapy.

DISCUSSERS: C. A. Hamberger, Göteborg, Sweden; Pedro Hernandez Gonzalo, Havana, Cuba; Eelco Huizinga, Groningen, Netherlands; Albert von Riccabona, Vienna, Austria.

Applications to present voluntary papers are being received regularly and anyone wishing to present such a paper should make known his intentions prior to the deadline of Oct. 1, 1956.

The motion picture sessions, as well as the scientific exhibits, will prove to be outstanding in every respect. It is essential that anyone wishing to present an exhibit should make his intentions known immediately, as the final date for consideration is Aug. 1, 1956; applications to present motion picture films should be sent in before Oct. 1, 1956. Anyone planning to attend the Congress and who has not yet registered should do so immediately in order to obtain hotel registration priority.

For more detailed information pertaining to the Sixth International Congress please communicate with the General Secretary, 700 N. Michigan Ave., Chicago 11, Ill., U. S. A.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this *information up to date*).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. John R. Lindsay, 950 East 59th Street, Chicago 37, Ill. ....
Vice-President: Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.
Secretary-Treasurer: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Statler Hotel, Washington, D. C., May 4, 1957.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. LeRoy A. Schall, Boston, Mass.
First Vice-President: Dr. Henry M. Goodyear, Cincinnati, Ohio.
Second Vice-President: Dr. Robert E. Priest, Minneapolis, Minn.
Secretary: Dr. Harry P. Schenck, Philadelphia, Pa.
Treasurer: Dr. Fred W. Dixon, Cleveland, Ohio.
Meeting: Statler Hotel, Washington, D. C., May 3, 1957.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Percy Ireland, Toronto, Canada.
President-Elect: Dr. Lewis F. Morrison.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Statler Hotel, Washington, D. C., May, 1957.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: John R. Lindsay, M.D., Chicago, Ill.
Vice-Chairman: James W. McLaurin, M.D., Baton Rouge, La.
Secretary: Hugh A. Kuhn, M.D., Hammond, Ind.
Representative to Scientific Exhibit: Walter Heck, M.D., San Francisco, Calif.
Section Delegate: Gordon Harkness, M.D., Davenport, Iowa.
Alternate Delegate: Dean Lierle, M.D., Iowa City, Iowa.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., Oct. 14-20, 1956.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Clarence W. Engler, 2323 Prospect Ave., Cleveland, Ohio.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia, Pa.
Meeting: Mark Hopkins Hotel, San Francisco, Calif., May 21-23, 1958.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill., October 8-11, 1956.

THE AMERICAN RHINOLOGIC SOCIETY

President Dr Ralph H Riggs [illegible] Shreveport La
Secretary Dr James Chessen [illegible] Denver Colo
Annual Clinical Session Illinois [illegible] Hospital Chicago Illinois October, 1956
Annual Meeting Palmer House Chicago Illinois October 1956

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

President Dr D M Lierle, University Hospital Iowa City Iowa
Secretary Treasurer Dr Michael H Barone, 168 Delaware Ave, Buffalo 2, N Y
Meeting Palmer House, Chicago Ill October, 1956

OTOSCLEROSIS STUDY GROUP

President Dr Gordon D Hoople, 1100 East Genesee St, Syracure N Y
Secretary Dr Lawrence R Boies University Hospital, Minneapolis 14, Minn
Meeting Palmer House, Chicago, Ill, October, 1956

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY

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Vice President Dr Kenneth Hinderer, 102 Medical Arts Bldg Pittsburgh, Pa
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PAN AMERICAN ASSOCIATION OF OTO RHINO-LARYNGOLOGY AND BRONCHO ESOPHAGOLOGY

President Dr Jose Gros, Havana, Cuba
Executive Secretary Dr Chevalier L Jackson, 3401 N Broad St, Philadelphia 40, Pa, U S A
Meeting Sixth Pan American Congress of Oto Rhino Laryngology and Broncho-Esophagology
Time and Place Brazil, 1958

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

President Dr Arthur W Proetz, Beaumont Bldg, St Louis, Mo
General Secretary Dr Paul Holinger, 700 No Michigan Ave, Chicago 11, Ill
Meeting Statler Hotel Washington, D C, May 5 - 10, 1957

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY

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Vice-President Dr John J O'Keefe
Treasurer Dr Joseph P Atkins
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Executive Committee Dr Harry P Schenck, Dr , Benjamin H Shuster, Dr William A Lell, Dr , William J Hitschler

BALTIMORE NOSE AND THROAT SOCIETY

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Secretary Treasurer Dr Theodore A Schwartz

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

President Dr Raymond Kerwin 310 So Michigan Ave, Chicago Ill
Vice President Dr Frank Wojniak, 6132 So Kedzie Ave, Chicago Ill
Secretary Treasurer Dr Stanton A Friedberg 122 So Michigan Ave, Chicago Ill
Meeting First Monday of each Month, October through May

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
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Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. J. L. Levine.
Vice-Chairman: Dr. Russell Page.
Secretary: Dr. James J. McFarland.
Treasurer: Dr. Edward M. O'Brien.
Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Frederick A. Holden, Medical Arts Building, Baltimore 1, Maryland.
Chairman-Elect: Dr. Sherman B. Forbes, 706 Franklin Street, Tampa 2, Florida.
Vice-Chairman: Dr. William J. G. Davis, 1632 K Street, N. W., Washington 6, D. C.
Secretary: Dr. G. Slaughter Fitz-Hugh, 104 East Market Street, Charlottesville, Virginia.

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Howard L. Mitchell, Lexington, Va.
President-Elect: Dr. L. Benjamin Sheppard, 301 Medical Arts Building, Richmond 19, Va.
Vice-President: Dr. Edgar Childrey, Jr., Professional Building, Richmond, Va.
Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Va.
Annual Meeting.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. H. K. Rouse, 1300 27th Ave., Gulfport, Miss.
Vice-President: Dr. A. J. McComiskey, 3420 Prytonia St., New Orleans, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting: The Edgewater Gulf Hotel, Edgewater Park, Miss., May 17-18, 1957.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. J. C. Peele, Kinston Clinic, Kinston, N. C.
Vice-President: Dr. George E. Bradord, Winston-Salem, N. C.
Secretary-Treasurer: Dr. J. D. Stratton, 1012 Kings Drive, Charlotte 7, N. C.
Meeting:

SOUTH CARO NA SOCIETY OF OPHTHALMOLOGY
D OTOLARYNGOLOGY.

President: Dr. Nor ddy, Sumter, S. Car.
Vice-President: D Gressette, Orangeburg, S. Car.
Secretary-Treasurer Roderick Macdonald, 333 East Main St., Rock Hill, S. Car.

FLORI CIETY OF OPHTHALMOLOGY
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President: Dr. Ch ace, 115 King St, St. Augustine, Fla.
President-Elect D Taylor, 706 Franklin St, Tampa, Fla
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THE GREATER M YE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. A der, 605 Lincoln Road, Miami Beach, Fla
Vice-President: D D Benton, Jr.
Secretary: Dr. Jam endel, Jr, 7241 Red Road, Miami, Fla
Meeting: Quarterly n Seas Restaurant, February, May, October, and December

DALLA DEMY OF OPHTHALMOLOGY
OTOLARYNGOLOGY

President: Dr. Edw rafton
Vice-President: Dr Pirchgott.
Secretary-Treasurer al W. Maxwell.

LOS ANG SOCIETY OF OPHTHALMOLOGY
OTOLARYNGOLOGY

President: Leland R e M D.
Secretary-Treasurer Rome, M.D
Chairman of Opthalm Section: Stephen J Popovich, M D.
Secretary of Ophthalm Section: Richard Kratz, M D.
Chairman of Otolaryngolo Section: Harold Boyd, M D
Secretary of Otolaryngolo Section: Howard G. Gottschalk, M D.
Place: Los Angeles C ty Medical Association Building, 1925 Wilshire Boulevard, Los Angeles California.
Time: 6:00 P.M, first Monday of each month from September to June inclusive—Otolaryngology Section. 6:00 P. M. first Thursday of each month from September to June inclusive—Ophthalmology Section.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: H. Leroy Goss, M D, 620 Cobb Bldg, Seattle 1, Washington
Secretary-Treasurer: Homer E. Smith, M D, 508 East South Temple, Salt Lake City, Utah.
Meeting: April 7-11, 1957

THE SOCIETY OF MILITARY OTOLARYNGOLOGISTS.

President: Col Wendell A Weller.
Secretary-Treasurer: Major Stanley H. Bear, M C, 3810th USAF Hospital, Maxwell AFB, Alabama
Time and place of meeting. October 16, 1956, Palmer House, Chicago, Ill

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Orrie E Ghrist, 210 N. Central Ave, Glendale, Calif
Treasurer: Dr. Norman Jesberg, 500 So Lucas Ave, Los Angeles 17, Calif
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Mid-Winter Clinical Convention annually, the last two weeks in January at Los Angeles, Calif.

[illegible] [illegible] ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

[illegible] Dr. [illegible] Bremerton, Wash.
[illegible] Dr. [illegible] Jensen, Seattle, Wash.
[illegible] Dr. [illegible] 1215 Fourth Ave., Seattle, Wash.

CANADIAN OTOLARYNGOLOGICAL SOCIETY SOCIÉTÉ CANADIENNE D'OTOLARYNGOLOGIE.

President: Dr. G. M. T. Hazen, 208 Canada Bldg., Saskatoon, Sask.
Secretary: Dr. G. Arnold Henry, 170 St. George St., Toronto, Ontario.
Meeting: Banff Springs Hotel, Banff, Canada, June 17-19, 1957.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Theodor Hunermann, Dusseldorf, Germany.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadephia 40, Pa., U. S. A.
Meeting: Sixth International Congress of Bronchoesophagology, Philadelphia, May 12-13, 1957.

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting: Lima, Peru, 1957.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

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Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salavador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

SOCIEDAD CUBANA DE OTO-LARINGOLOGIA.

President: Dr. R[illegible] de Villiers.
Vice-President: [illegible] de Cár[illegible]s.
Secretary: Dr. [illegible]

SOCIE[illegible] DE LA HABANA.

Presidente: Dr.
Vice-Presidente:
Secretario: Dr.
Tesorero: Dr.

MEXIC[illegible] [illegible]RGE

[illegible] Dr. C
[illegible]t: D
Dr. J

SC

BUENOS AIRES CLUB OTOLARINGOLOGICO.

Presidente Dr K Segre
Vice Presidente Dr A P Belou
Secretario Dr S A Aranz
Pro Secretario Dr J M Tato
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SOCIEDAD COLUMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLUMBIA).

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Secretario Dr Felix E Lozano
Tesorero Dr Mario Arenas A

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA

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President Dr Salvador Mixco Pinto
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SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA

Presidente Dr D Adolfo Hinojar Pons
Vice Presidente Dr D Jose Perez Mateos
Secretario General Dr D Francisco Marañés
Tesorero Dr D Ernesto Alonso Ferrer

SOCIEDAD OTO-RINO LARINGOLOGIA DE LOS HOSPITALES DE MADRID

Presidente Dr Don Fernando Beltrán Castillo
Secretario General Dr Don Alfonso Vassallo de Mumbert
Tesorero Dr Don Rafael García Tapia

PORTUGUESE OTORHINOLARYNGOLICAL SOCIETY.

President Dr Albert Luis de Mendonca
Secretary Dr Antonio da Costa Quinta, Avenida, de Liberdale 65, 1° Lisbon

SOCIEDADE PORTUGUESA DE OTORRINOLARINGOLOGIA E DE BRONCO ESOFAGOLOGIA

Presidente Dr Alberto Luis De Mendonca
Vice Presidente Dr Jaime de Magalhaes
1 ° Secretario Dr Antonio da Costa Quinta
2 ° Secretario Dr Albano Coelho
Tesoureiro Dr Jose Antonio de Campos Henriques
Vogais Dr Teofilo Esquivel
Dr Antonio Cancela de Amorim
Sede Avenida da Liberdade, 65, 1°, Lisboa

various factors responsible for production of this annoying symptom, such as dry central heating, tobacco, alcohol, dust and fumes, allergies, fungous irritations, atrophy, metabolic and endocrine disturbances and prolonged medication, were discussed. Because postnasal discharge represents one of the commonest complaints of patients seen by otolaryngologists, all practicing this specialty should read this article, which is probably the best on this subject to come under our observation.

Neistadt[17] reported results of a study of a new sympathomimetic agent, tetrahydrosoline hydrochloride, as a nasal decongestant solution (0.1 per cent) in 204 patients (12 with allergic rhinitis and 192 with nasopharyngitis or sinusitis or both). All but one patient, whose response was only partial, reported excellent results. In addition to its promptness of action, degree of relief provided and percentage of patients benefited, it displayed prolonged action and no untoward local or systemic effects.

Weinstein and Sachs[18] reported a case of a 70-year-old woman with pemphigus of the mouth, oropharynx and larynx without cutaneous manifestations. Since primary pemphigus involving only these areas is uncommon, the case is described in detail. Because of laryngeal stenosis caused by the pemphigus, tracheotomy was required. These authors briefly reviewed the literature and discussed the micropathology of the lesion.

Hoover[19] discussed his general observations and experiences at the Lahey Clinic on the functional abnormalities of the pharynx and their sequelae. He described dysphagia due to vagus nerve paralysis and illustrated this with case reports. This leads into the subject of pulsion diverticula of the pharyngoesophagus and the accompanying spasm or stenosis of the cricopharyngeus. Cricopharyngeus relaxing procedures and plastic reshaping of the hypopharynx are described in the case of vagus paralysis, as well as pulsion diverticulum. Unlike Lahey, who preferred the two-stage diverticulectomy, Hoover considered the single stage procedure preferable. The latter portion of this article deals with general considerations of esophagoscopy.

Lindsay[20] reviewed the various neurologic, psychologic and structural disturbances that might cause functional difficulties of the upper swallowing mechanism. In a general discussion of the swallowing mechanism, he pointed out that its direct action cannot be observed, but fluoroscopy and roentgenography can provide useful objective evidence. Disturbances due to poor muscular action, interrupted innervation, and psychomotor influences are discussed. The article is illustrated with appropriate roentgenograms. Roentgenography can confirm weakness or asymmetry of action of the constrictor muscles and of the cricopharyngeus commonly seen in disturbances of musculature and innervation, but present techniques cannot demonstrate evidence of increased tone or spasm of these muscles.

Benedict and Sweet[21] reported two cases of dysphagia due to hypertrophy of the cricopharyngeus muscle or hypopharyngeal bar. They quoted Templeton and Kreider, who stated that the cricopharyngeus muscle is a true sphincter which can be demonstrated roentgenologically. When the Valsalva test is performed, contraction of the cricopharyngeus is intensified. Brunner and Sweet stated that the purpose of the tonic contraction of the cricopharyngeal sphincter is to prevent esophageal respiration. Relaxation of the cricopharyngeal sphincter would cause air to be sucked more readily into the esophagus than into the lungs. The mound on the posterior pharyngeal wall at the level of the cricopharyngeus muscle may be called the "hypopharyngeal bar," and this indicates spastic contraction of the cricopharyngeal muscle. In the two cases of dysphagia, reported by Benedict and Sweet, hypertrophy of the cricopharyngeus muscle was demonstrated roentgenologically, and since all other causes of dysphagia were eliminated, it was concluded that the presence of a hypopharyngeal bar was responsible for the existing dysphagia.

DIVERTICULA.

A good short general review of diverticula of the upper esophagus, without author credit or references, appeared in the *Medical Times*[22]. No cases or statistics were included.

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In a brief but excellent article Neville[23] cautioned that although most pharyngo-esophageal diverticula can easily be handled through a cervical approach, occasionally one may encounter an extremely large diverticulum projecting into the thorax. In such cases it may be judicious to open the thorax and free the diverticulum from continuous intrathoracic structures, after which the usual surgical approach can be employed and the diverticulum easily lifted into the neck. Such a case was encountered by Neville. Because practically all pharyngoesophageal diverticula measure less than 5 cm., most of them can be removed without difficulty through the usual cervical incision; however, considerable difficulty may be experienced in attempting to remove extremely large ones through such an incision because of the proximity of major vessels and the possibility of injury to these structures. The thoracic approach minimizes these dangers by freeing the diverticulum from the proximity of these structures.

In a discussion of pharyngo-esophageal diverticulectomy Jackson and Norris[24] called attention to the fact that approximately 75 per cent of their cases occurred in males, but offered no explanation for this. Although it is generally accepted that pharyngo-esophageal diverticula occur only in elderly people, two cases are reported in individuals 21 and 27 years of age. The surgical technique employed is identical with that described by other surgeons, and in keeping with the present day trend, they preferred the one stage technique. Esophagoscopic guidance in determining amputation of the neck of the diverticulum is advocated. The one complication of stenosis in their series occurred in a patient in whom esophagoscopic guidance was not used. Having experienced the same complication under identical conditions as reported by Jackson and Norris, we heartily concur that esophagoscopic guidance is highly desirable during amputation of the neck of the diverticulum.

A brief review of the problem of pulsion diverticulum of the upper esophagus is offered by Howden[25]. He presented reports of nine cases in which single-stage diverticulectomy was performed and gave a detailed outline of the surgical technique used.

Johnson[26] believed that the otolaryngologist is unnecessarily depriving himself of a stimulating field of surgery, namely, repair of pulsion diverticulum of the hypopharynx. He stated that most recent references to this lesion are found in the general surgical literature, despite the fact that the anatomic and surgical features clearly fall within the practice of the otolaryngologist, who is interested in surgery of the neck Johnston covered the subject well in a short discussion, and included a case report.

Fox and associates[27] apparently considered pharyngeal diverticulectomy a procedure for a relatively uncommon condition. They presented the first such case seen at the Kent General Hospital. Nothing new or constructive is offered in reporting a routine case. It is surprising to read that a stump remained after excision of the diverticulum and that the opening into the hypopharynx was closed with suture material as large as chromic No. 1. This, coupled with the fact that phenol and alcohol were applied to the stump, is reminiscent of a general surgeon performing an appendectomy. The authors reported that the diverticulum developed on the lateral wall of the hypopharynx, whereas it has been established that all such diverticula invariably develop in the median line of the posterior wall and gradually develop to one side or the other, most frequently to the left side.

Treatment of traumatic perforations of the esophagus has undergone considerable change in the last two decades. Prior to 1935 uncontrolled mediastinitis and death followed esophageal perforations in 70 per cent of the cases unless surgical drainage was instituted. Kesseler and Maier[28] stated that early recognition and prompt treatment of traumatic perforations of the esophagus are essential to preserve life Although the early use of antibiotics may lower the mortality rate considerably, immediate surgical intervention is usually necessary Kesseler and Maier reported a case of perforation in the lower portion of a diverticulum Surgical drainage was instituted by performing right cervical mediastinotomy; however, the exact location of the perforation was not initially known When temperature persisted, a search revealed the location of the perforation and diverticulectomy was performed with beneficial results.

ROENTGENTHERAPY.

Reich[29] emphasized the fact that radiologists have long recognized that lymphoid tissue is the most sensitive of normal structures in the human body, to radiation. Recognizing the value of the Crowe radium applicator as used in the nasopharynx, and its limitations, Reich conducted a study of 105 patients referred for roentgentherapy to the nasopharynx. Of these 68 were available for follow-up study. The ages of the patients ranged from 18 months to 55 years. Thirty-eight with hypertropied lymphoid tissue in the nasopharynx complained of loss of hearing. After treatment 28 patients showed improvement. Good results were obtained in five patients with soft tissue masses in the nasopharynx treated by radiation. Other types of cases were treated successfully so that Reich was forced to the conclusion that roentgentherapy in small doses is a useful adjunct for treating hypertrophied lymphoid tissues in the nasopharynx, especially if the presenting symptom is deafness.

Green and associates[30] presented an excellent discussion of the indications for and technique of irradiation of the Eustachian tubes for the reduction of lymphoid tissue that is blocking adequate drainage of the middle ear. They sent questionnaires to the parents of 165 children treated by external Roentgen-ray irradiation during a period of five-and-one-half years. The answers received would indicate less hearing loss, less mouth breathing and snoring, and less respiratory infections following Roentgen therapy. It should be noted, however, that no control group of untreated patients was included for comparison.

An editorial[31] in the *British Medical Journal* reviewed the works of Crowe, Guild, Bordley and Hardy on the effects upon hearing defects of radium therapy to the nasopharynx of children. The hope was expressed that further investigations on this subject might follow.

Van Herik and Erich[32] devised a bakelite applicator for radium therapy to the nasopharynx. The technique for use of the applicator was described and well illustrated. They used this technique to supplement Roentgen therapy for squamous cell carcinoma of the nasopharynx in 30 patients without untoward complications

SURGERY

A number of aspects of the subject of tonsillectomy was discussed by Moore[3], in the light of his experience He reviewed the subject of tonsillectomy with use of a general anesthetic, and described his technique of peritonsillar injection of a vasoconstricting solution to facilitate the procedure and to limit loss of blood There was a brief discussion and warning about the use of efocaine "R" for prolonged relief of pain following tonsillectomy Moore concluded with a review of possible untoward and possible beneficial effects of ACTH and Cortisone as related to the problem of tonsillectomy performed with use of a general anesthetic This is a timely and interesting article on an old subject

Marshall and associates[34] were favorably impressed with Kutapressin, a non-toxic, selective vasoconstrictor, in the prevention of hemorrhage following tonsillectomy and adenoidectomy No scientific information concerning the chemical or pharmacologic nature of the product was furnished, nor did they mention animal experiments to determine possible toxic qualities of the drug No control series of cases was presented, and no factual information was given concerning the 100 cases mentioned

A questionnaire was mailed by Hamrick[35] to every hospital in the United States approved for residency training in otolaryngology and anesthesiology in an effort to study anesthetic preferences for tonsil and adenoid operations in children The results from 155 hospitals were tabulated The first choice was vinyl ether induction with ether maintenance by insufflation Hamrick listed the first ten choices and stated that endotracheal techniques are increasing in popularity, owing to their well recognized margin of safety in the hands of the well trained anesthetist

A study of the effects of depot local anesthetics injected into the operated area after tonsillectomy was made by Reed and Montgomery[3] at the Massachusetts Eye and Ear Infirmary. They used efocaine in their study. Of the 90 patients, only 27 received the drug; the remaining 63 served as controls. They concluded that this drug has no significant effect upon the severity or duration of the postoperative discomfort or complications.

Folds and pockets in the nasopharynx are observed surprisingly often in routine nasopharyngeal examinations. Tolczynski's[37] investigations revealed their incidence to be 47.5 per cent, and the nasopharynx presented a variety of strings, folds, bands, crypts and pockets which varied with each patient. Each of these is illustrated and described in detail. Tolczynski stated that the residual masses, after a faultily performed adenoidectomy, are more susceptible to infection than the intact adenoid. Deep folds and pockets in the nasopharynx are largely responsible for annoying postnasal drip due to chronic infection. Adenoidectomy under direct vision with use of a general anesthetic is the method of choice in the prevention of formation of folds, crypts and pockets. This is an interesting and illuminating article that should be read by all otolaryngologists performing adenoidectomies.

BENIGN TUMORS.

Surgical removal of benign nasopharyngeal tumors frequently taxes the ingenuity of the surgeon. The inaccessibility of the nasopharynx and the vascularity of the lesions arising in that area are factors which must be overcome for successful management of these tumors. White[38] pointed out that the principal benign tumors found in this area are nasopharyngeal fibromas and large choanal polyps. Removal of the former type presents a formidable problem at times because of severe bleeding. White described various approaches and techniques advocated for removal of these tumors, and described a combined intranasal and postnasal approach that he devised and used successfully for removal of fibromas. It consists in passing a loop of wire through the nose into the oropharynx, and with appropriate palate retractors, placing the wire loop around the tumor. An insulated rigid tube protects the nasal mucosa while a cautery tip makes contact with the wire. Pulling down on the wire loop resects the tumor as closely to its base as possible with minimum bleeding.

Mills[39] reported a case of midline cyst of the nasopharynx which could be seen protruding a few millimeters below the level of the uvula. A preliminary diagnosis of polyp was made, and a transpalatal approach was used to expose the cyst to better vision. The dome of the cyst was excised by diathermy and a small blind pit was exposed. It was concluded that this cyst was formed from the vestigial remnants of Rathke's pouch.

We are reminded by Beales[40] that simple tumors of the pharynx are uncommon. Probably the commonest of these is the "mixed salivary tumor." It arises from the three pairs of salivary glands, as well as from salivary gland tissue regardless of its location. Thus, these tumors arise from the small glands that are widely situated throughout the oral cavity in the lips, cheek, palate and floor of the mouth. The serous cell is probably more prone to metaplasia than the mucous type; therefore, salivary tumors are commoner in the parotid gland. Beales reported an interesting case diagnosed as a peritonsillar abscess which proved eventually to be a "mixed salivary tumor." An attempt was made to remove the tumor intra-orally; but the futility of this approach was quickly realized, and the operation was terminated. Later through an external approach the tumor was removed without difficulty. The unsuccessful original operation emphasizes the fact that sizable tumors should never be approached intra-orally, as they are not only difficult to dissect, but control of hemostasis may become such a problem as to place the patient's life in jeopardy.

Two interesting and unusual cases of plasmacytoma originating in the nasopharynx are presented by Emslie-Smith and co-workers[41]. In both cases the initial lesion was a soft tissue plasmacytoma of the nasopharynx and adjoining areas, followed by appearance of widespread cutaneous plasmacytoma and involvement of bone and lymph nodes. Extramedullary plasmacytomas are uncommon tumors, affecting males more frequently and generally occurring in the mucous membranes of the upper air passages. Their histologic appearance gives little clue to their future course which varies from complete recovery with treatment to the development of myelomatous lesions throughout. As a rule these lesions respond well to irradiation early in the illness, but later are more resistant.

Mixed tumors in the orofacial region are common, but mixed tumors located specifically on the posterior pharyngeal wall are rare. Havens and Butler[12] reviewed 41 cases encountered at the Mayo Clinic among 1,500,000 clinic registrations during a period of 37 years. After reviewing the general subject of mixed tumors and commenting on methods of treatment in the past, they discussed the surgical approaches used in their 41 cases. They believed that an external approach is advisable in some cases of pharyngeal mixed tumor. Their follow-up has been good and their recurrence rate low. They observed that there seemed to be less "seeding" in the wound after internal excision than after the external incision. They believed that this might be explained by the fact "that a clean surgical wound that is closed, as a neck wound is closed, would provide much better soil for possible 'seeding' than would a wound which is packed wide open with iodoform gauze and which is left in place for four to seven days."

Epstein and Loeb[13] reported an unusual case, in an infant three-weeks-old, in whom a thymic tumor was found in the hypopharynx. The original discovery was made by the baby's mother, who noted what appeared to be a second tongue when the infant regurgitated. No difficulty was encountered in its removal and the pathologist rendered a report of hamartoma of the pharynx. Epstein and Loeb reminded us that the presence of an aberrant thymus above the level of the thyroid gland is rare. After extensive study they concluded that this thymic tumor was probably of embryologic origin from the fourth visceral cleft.

A general review of juvenile fibromas of the nasopharynx by Erich[14] emphasized the dangers involved in biopsy of these lesions. Erich believed that biopsy should not be performed if the diagnosis is obvious clinically, and if biopsy is necessary it should be done only in the hospital where adequate precaution can be taken to control hemorrhage. The pathology, etiologic factors, symptoms and treatment were reviewed. Erich's present choice of therapy is implantation of radon seeds repeatedly every three to six months until the tumor has disappeared. The specific technique for radon implantation was described.

MALIGNANT TUMORS.

Duboe[15] stressed the importance of examination of the nasopharynx of each new patient coming to the office, particularly if the patient's symptoms suggest nasopharyngeal disease. He properly emphasized the fact that practically every nasopharynx can be examined adequately if the examiner is willing to take sufficient time to carry out this examination. Malignant tumors of the nasopharynx are more common in this area than are benign tumors, and the usual sites for these malignant lesions are the fossa of Rosenmuller, posterior wall on either side of the midline and roof of the nasopharynx. Nasopharyngeal cancer is relatively common in China, and in Hong Kong it is reported to be the second commonest malignant disease. The prognosis is unfavorable even though the lesion is discovered early. Sarcomas offer a more favorable outlook than do carcinomas. Malignant lymphoma, reticulum cell sarcoma, lympho-epithelioma, and transitional cell carcinoma respond well to irradiation, but the rate of recurrence is high. Involvement of the cranial nerves makes the prognosis more unfavorable and metastasis is usually limited to the cervical nodes.

In an excellent presentation Missal[16] discussed the problem of adequate surgical treatment of extensive carcinoma of the pharynx, larynx and cervical esophagus, which he considered to be one of the most distressing clinical problems encountered by the laryngologist. In the past most of these neoplasms have been considered inoperable and irradiation has been the principal therapy offered. The benefits achieved in these cases by irradiation leave much to be desired. For this reason some of those lesions are now being excised with encouraging results. Missal did not use the term radical surgery in these cases but preferred using the designations of adequate and inadequate surgery. Use of these terms is predicated upon the existence of three factors: first, the surgeon must possess sufficient skill and knowledge to perform an adequate operation; second, he must execute the required surgical procedure, and third, he must exercise sound judgment in the selection of patients suitable for the operation. A historical review, as well as critical comments on cases presented, adds to the interest of the presentation. Surgical technique is discussed and emphasis is placed on preoperative preparation of the patient with proper attention to fluid and electrolyte balance. Although this presentation includes the pharynx, it is principally concerned with surgical treatment of malignant lesions of the larynx, pyriform fossa, mouth of the esophagus and with neck dissection. Since the present review encompassses only pharyngeal surgery, this most excellent presentation is briefly sketched and those interested should read the entire article.

In a review of the treatment of hypopharyngeal carcinomas Morfit[47] advocated laryngopharyngectomy and simultaneous block dissection of the neck as the treatment of choice. He concluded that other forms of therapy have not yielded good statistical cure rates, and believed that wide dissection would improve the incidence of cures. His discussion included an illustrated operative description and details of postoperative problems. No case reports or statistical studies were offered in support of the opinions expressed.

Jack[18] discussed the surgical problem that carcinoma of the hypopharynx and upper esophagus presents. He believed that wide resection of the tumor with immediate reconstruction of the area and restoration of swallowing should be the desired objective. He reported the case of a postcricoid lesion with involvement of the esophagus from the level of the cricoid for a distance of about 6 cm. Left postero-lateral thoracotomy through the bed of the resected eighth rib offered an approach to the esophagus and stomach. After the operation was completed in this area, the patient was placed on his back and laryngo-pharyngectomy and partial esophagectomy were performed. The lower portions of the esophagus and stomach were brought up and the upper portion of the esophagus was sutured to the pharynx (pharyngo-esophageal anastomosis). The author described an interesting and formidable surgical procedure, but failed to include follow-up notes or discuss prognosis.

Martens[49] reported what he believed to be the first recorded case of malignant synovioma in the hypopharynx. A photograph of the gross specimen and a diagrammatic drawing were shown. Five cases of benign and malignant synovial tumors from other regions of the body were also described, and the general pathology was briefly discussed.

Green[50] discussed the mutual invasion of two independent tumors occurring simultaneously in the same pharynx. One was a squamous cell carcinoma of a tonsil, and the other a fibrosarcoma of the lateral pharyngeal wall. Where the two had been in opposition to each other, mutual implants had occurred at the site of collision. This paper was written by a pathologist and is well documented by photomicrographs of the tumors.

Sanghvi and coworkers[51] conducted an investigation on the smoking and chewing of tobacco in relation to cancer of the upper alimentary tract. Their studies showed that intraoral cancer is relatively more prevalent in Bombay than in New York or London. Even in regard to the commonest site of oral cancer there is a noticeable difference in various localities in India. Cancer of the lip is the commonest type of cancer in Bihar; of the buccal mucosa and alveolar margin in Travancore and some parts of Madras; of the base of the tongue in Bombay; of the oral surface of the palate in Andhra, and of the hypopharynx in Assam. Personal history and habits relative to alcohol, smoking and chewing were studied carefully, and the results of their investigation showed that the habit of chewing was associated with cancer of the oral cavity; that the combined habit of smoking and chewing was associated with cancer of the hypopharynx and base of the tongue; and that smoking alone was associated with cancer of the oropharynx and esophagus.

The surgical treatment of extensive cancer of the cervical esophagus, pharynx and larynx, when it has been necessary to remove the total circumference of the swallowing tube, has been encumbered by the delayed multiple stage reconstruction of the pharynx and esophagus by use of transposed tissue flaps from the neck or chest. Conley[52] described an operation designed to combine the excisional and reconstructive stages into a single composite one stage procedure. After radical resection of the cancerous growth, the continuity of the pharynx and cervical esophagus is restored by implantation of a thick, split free skin graft draped over a woven nylon tubular stent which is carefully sutured to the mucous membrane. Conley described and illustrated the technique for this all-important surgical procedure, which undoubtedly represents the latest advance in reconstructive surgery for such cases.

The twelfth reported case of carcinoma occurring in a pharyngeal or esophageal diverticulum was presented by Riberi, Battersby and Vellios[53]. They stressed the diagnostic importance of a sudden loss of weight and a change in character and severity of dysphagia in long standing cases of pharyngoesophageal diverticula, as evidence of possible malignant lesions. They successfully treated their patient, a woman 82 years old, by surgical excision of the diverticulum with its carcinoma, and they advocated this therapy for all such cases in which the malignant growth is still localized to the sac.

Batsakis and associates[54] reported a case of nasopharyngeal angiofibroma containing in its center a fibrosarcoma. The patient was 54 years of age at the the time of definitive surgical removal of the tumor and was 48 years of age at the time of the original diagnosis of angiofibroma. Several attempts at removal were made during this five year period, and extensive radiation therapy was then given. The tumor continued to be considered benign pathologically until after radiation therapy. Final excision 11 months following irradiation produced a specimen in which the malignant changes were found. The relation of these changes to "radiation cancer" was discussed briefly.

The origin of nasopharyngeal chordoma was briefly described by Crikelair and McDonald[55]. A nasopharyngeal chordoma is a slowly growing tumor that invades and destroys locally. Radiation therapy will result in a temporary favorable response in some cases and subtotal removal of large tumors may prolong life by relieving compression. A case is reported of a white boy in whom symptoms referable to a nasopharyngeal tumor developed at the age of 11 years.

Radiation therapy, radium needles and surgical treatment have made it possible to keep this patient alive for eight years (at the time of the report the patient was 19 years old). The accompanying photographs show massive facial distortion before treatment and marvelous results following plastic surgery.

Lawley and Mekie[56] studied the effects of urethane and triethylene melamine (TEM) on separate series of patients with nasopharyngeal malignant lesions. Temporary palliative effects were noted in 12 of 16 patients who completed a full course of treatment with urethane. They believed that the possibility of using urethane as a form of palliative therapy should be considered when: *a*, radiotherapy is not available; *b*, the disease is too advanced for radiotherapy; and *c*, there is an interval before radiotherapy can be begun. No significant improvement could be attributed to TEM in the seven patients treated. Leukopenia and pronounced fall in the number of granulocytes developed after administration of the drug.

St. John and Woo[57] commented on the rarity of rhabdomyosarcomas, and further pointed out that these tumors in the nasopharynx occur chiefly in childhood or adolescence, and at first have the appearance of nodular polypoid tumors which are white or flesh colored, depending upon the amount of vascularization. They reported such a case in a four-year-old boy. The nasal passage was completely filled with a polypoid-like tumor. A mass could be seen protruding from behind the uvula. This polypoid tissue was removed by means of a nasal snare, and a diagnosis of rhabdomyosarcoma was established. Roentgen therapy controlled growth of the tumor only temporarily.

A case of dilatation of the pharyngo-esophagus following total laryngectomy was reported by Cracovaner and Rubenstein[58]. The dilatation was so great that it interfered with deglutition and esophageal speech. They discussed possible mechanisms of formation and described a method of repair.

Ashley, Briggs and Affley[59] reviewed the histories of four of their patients requiring reconstructive surgery of the pharynx following radical excision for carcinoma. Each case illustrates a different technique and type of repair. Photographic and diagrammatic illustrations greatly aid the descriptive text. Of special interest is the first case, in which primary closure of a strip of remaining pharyngeal mucosa was accomplished over a gastric feeding tube. Then in two days the gastric tube was removed, and a feeding gastrostomy was performed. Liquids were begun by mouth 18 days postoperatively. No details were given concerning the rationale for removing the nasogastric tube and maintaining nutrition through a gastrostomy.

A clear concise discussion of wide surgical excision of carcinoma of the hypopharynx was offered by Snitman and Horwitz[60] as a preliminary report. An implied promise of follow-up studies, as well as a definite statement that a paper is being prepared concerning reconstructive phases of the work, adds to the depth of the paper. These authors classified carcinomas of the hypopharynx into three groups, depending upon gross spread of the primary lesion. They believed that operation is indicated in all cases in which the malignant tissue can be removed *en bloc.* They considered reconstructive techniques to be adequate to make even complete excision of the pharyngeal mucosa feasible, even though no pharyngeal mucosa be left to close or to participate in future closure. Their early cure rate has been better than in any other type of therapy. A five-year analysis is not yet available.

Moore and Martin[61] reviewed all cases of primary melanoma of the oral cavity and upper respiratory tract that occurred at the Memorial Center in New York between 1930 and 1948. They found records of 26 cases that they considered acceptable. There were four cases of melanoma of the oropharynx; all were limited to the palatine tonsil. Only one patient had initial symptoms related to the primary site, whereas the other three had initial symptoms from metastatic lesions. Two of the four primary lesions were non-pigmented. None of the patients was given therapy directed toward a cure. Melanomas in other areas, such as the larynx and sinuses, were included.

After discouraging results with other methods of pharyngeal and upper esophageal reconstruction, Wilkins[62] began to employ a new method, utilizing the laryngeal and upper tracheal mucous membrane with the anterior portion of the laryngeal cartilages. He dissected the mucosa off the anterior face of the posterior half of the cricoid, if the carcinoma were postcricoid, and used this mucosa for reconstruction. He believed that the barrier of the cricoid cartilage should be adequate to prevent involvement of this anterior mucosa by the disease. The technique as used in two cases, was described and illustrated in detail. This article is recommended reading for all otolaryngologists interested in cancer surgery of the neck.

Rosenbaum and Seaman[63] called attention to the general failure to appreciate the neurologic manifestations associated with nasopharyngeal tumors. Their statement that intracranial extension is the earliest presenting sign of nasopharyngeal malignant tumors can easily be challenged. The anatomic location of the tumor favors early intracranial extension and neurologic signs particularly because of the proximity of the foramen lacerum and ovale and the IIIrd, IVth, Vth and VIth cranial nerves. The commonest initial symptom produced by these tumors, steady unilateral facial or aural pain, should demand thorough nasopharyngeal examination. Obstruction of the Eustachian orifice, blood streaked sputum or cervical adenitis are some of the other presenting symptoms. As the tumor grows larger, it will produce neurologic symptoms by invasion of cranial nerves. Unfortunately, however, in our experiences, tumors in this area are frequently not diagnosed early because they produce few symptoms in the early stages. We have found neurologic symptoms as late indications of tumors of the nasopharynx.

In a general discussion, designed for general physicians, Sebai[64] discussed trends in treatment of cancer of the lip, tongue, oral cavity, pharynx, tonsils and larynx. He concluded that the trend is toward more and earlier radical excision, and that this attitude is becoming noticeable even in countries, such as Sweden and France, where irradiation therapy has generally had its best results. The reasons for this apparent acceptance of radical excision are the advances in anesthesiology, antibiotics, blood banks and plastic repair. These advances permit more meticulous and time consuming surgical procedures than was formerly possible, and the cure rate has therefore been better. The figures of the Memorial Cancer Hospital of New York during the past ten years strongly substantiate this tendency. Several pages of photographs of the author's patients, who had undergone radical excision in the bucco-pharyngeal area, are included in an extremely readable paper.

In an excellent article on the indications and technique for cervical node resection Ochsner[65] discussed at length the problems confronting the surgeon when dealing with carcinomas of the lip, gingiva, tongue, floor of the mouth, soft palate, tonsil and pharynx. In the discussion of each of these the advisability of radical neck dissection was given due consideration; prophylactic radical resection was also discussed. Carcinoma of the tonsil represents about 10 per cent of all buccal cancers and involvement of lymph nodes is frequent and early. The treatment advocated for carcinoma of tonsils consists of intense irradiation followed by *en bloc* excision of the original lesion and unilateral radical neck dissection. This offers the patient the best chance of survival. Cancer of the nasopharynx is usually a highly malignant tumor and may be either epithelial or mesothelial in origin. Lymph nodes become involved early because of the rich lymphatic drainage in the region of the nasopharynx. Treatment consists in intensive radiation. Ochsner then described his technique of radical lymph node dissection of the neck. This is a most excellent article which should be read by all doing this particlar type of work. Lymphatic drainage from the various malignant areas is clearly and adequately illustrated, showing the location and the frequency of involvement of the various lymph nodes of the neck.

In an interesting and extremely readable paper on a subject that is receiving increasing attention, Hadfield[66] reported his methods and results in 24 surgically treated patients with cancer of the nasopharynx. His main concern is with successful and early closure of the pharynx, and he discussed the Wookey repair and several modifications. His best results were obtained by use of a combination pedicle graft and Thiersch graft, followed by secondary closure. Those readers interested in this subject should read the article and study the diagrams in order to appreciate fully the thoughts expressed

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MEDIASTINAL EMPHYSEMA COMPLICATING TRACHEOTOMY AND THYROIDECTOMY.*

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Mediastinal emphysema, or pneumomediastinum, is defined *as the presence of air in the mediastinum. Although not com*mon, its occurrence as a complication of operations about the neck is frequent enough to warrant an alert awareness of the possibility of its development in these situations. A thorough understanding of the mechanism of its production and an adequate appreciation of its clinical significance is essential for the intelligent management of these cases. The early recognition of its presence and the institution of prompt and appropriate measures to control it are important, and may be lifesaving.

Air may gain access to the mediastinum by several anatomic routes:

1. Directly through a perforation of the trachea, bronchi, or esophagus.

2. By dissection along the fascial planes of the neck.

3. From the interstitial tissues of the lung, by travelling to the hilus along the perivascular and peribronchial sheaths following rupture of pulmonary alveoli.

4. From the retroperitoneal space.

It is also theoretically possible for mediastinal emphysema to develop by the spread of air between the parietal pleura and the chest wall, or beneath the visceral pleura, or by passage of air from the pleural cavity to the mediastinum. Finally, artificial pneumomediastium may be deliberately induced by direct needle puncture, as originated for diagnostic purposes by Condorelli[1] in 1936.

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In mediastinal emphysema complicating operations on the neck such as tracheotomy and thyroidectomy, the only pathways requiring consideration are: direct perforation of the airway, spread along the fascial planes of the neck, and through the mechanism of interstitial pulmonary emphysema.

A review of the increasing number of case reports of this complication reveals a great variance in opinion among authors as to the mechanism involved.

Historical Aspects.

Emphysema in that form in which gas or air becomes collected under the skin or in the connective tissue was well known to Paul of Ægina[2] in the Seventh century A. D. There is little doubt that gas gangrene was included in this category, and it was not until the time of Ambroise Paré[3] in the Sixteenth Century that a more accurate concept of aberrent air in the subcutaneous tissues after trauma was developed; however, the principle of negative intrathoracic pressure was unfamiliar to these observers, and no notice was taken of the effect of trapped air within the thorax in patients exhibiting surgical emphysema with chest injuries. In 1767, William Hewson[4] reached the important conclusion that air is contained between the pleural layers in pneumothorax, and he decided it could be the cause of deleterious pressure symptoms even capable of producing death. His interest had been aroused by several reports[5,6,7,8] of chest wounds complicated by subcutaneous emphysema. In the light of the new knowledge gained from his experiments, he reevaluated these cases that had been erroneously interpreted, thereby gaining for them a niche in any history of this subject.

In 1826 Laënnec[9] published the second edition of his classical treatise on diseases of the lung, in which he accurately described the clinical condition of pulmonary interstitial emphysema, so frequently the precursor of mediastinal emphysema. In this great work, he displayed familiarity with certain aspects of its pathogenesis and with many of its symptoms and signs.

The clearer perception of pulmonary disease and the novel methods of physical examination of the chest introduced by

Laennec stimulated fresh interest in pathology of the lungs. This manifested itself, particularly in France, in a series of reports[10, 11, 12, 13] of clinical cases exhibiting the three stages of pulmonary, mediastinal and subcutaneous emphysema, and the correlation of them all as phases of a single entity termed generalized emphysema.

The observations of these clinicians were based almost entirely on patients with medical diseases associated with severe coughing, or on those with conditions accompanied by intense straining, as in parturition (Menier e's three patients).[10] The appearance of emphysema was readily and satisfactorily explained by Laennec's "Emphyseme Interlobulaire du Poumon" and its sequelae. From their experience it was believed that this complication in itself was not dangerous, and Laennec's statement that he had not seen anyone die of this condition alone was cited by Roger[13] in 1862 in support of this conviction ("Je n'ai vu mourir personne de cette affection seule").

Meanwhile surgeons were encountering cases of mediastinal emphysema associated with chest injuries, in which the mechanism appeared to be identical with those of medical origin. Dupuytren[14] regarded the infiltration of air into the mediastinum in traumatic cases with extreme gravity because of the presumed pressure on vital organs, and he attached an unfavorable prognosis to its advent. The whole subject received careful consideration by Demarquay[15] in his monograph published in 1886. He disagreed with Dupuytren's pessimistic view of mediastinal emphysema, because he did not believe sufficient pressure could be built up to affect seriously the important mediastinal organs when an easy escape route was present into the neck and subcutaneous tissues where the emphysema usually reveals itself first. He conceded, however, that in general the complication in surgical cases should be taken seriously, not because of any influence it had on the prognosis but as an indication of the gravity of the primary lesion causing it. He considered spontaneous emphysema, or that following effort, as not serious and as always terminating in cure.

Both Guillot[11] and Ozanam[1] had studied their autopsy material, and Rokitansky[16] completed Laennec's picture by describing the pathological aspects in his text book on pathology.

in 1856. Following this, in 1888 Müller[17] summarized the course of events from rupture of the pulmonary alveolar walls to interstitial emphysema of the lungs, thence to invasion of the mediastinum with air and finally to infiltration of the subcutaneous tissues of the neck to produce generalized emphysema. In addition, he noted the diminution of the area of cardiac dullness in mediastinal emphysema and described crepitant sounds dependent upon the heart beat. This report exhibited a comprehension of mediastinal emphysema approaching the modern concept.

In pursuing the development of this theory which eventually became known as the intrinsic mechanism, two more recent important contributions must still be given consideration. Through his clinical studies of mediastinal emphysema of apparently spontaneous origin, full credit is due to Hamman[18] for elucidating the diagnostic aspects of interstitial pulmonary emphysema and its sequel, mediastinal emphysema. The pathognomonic sign of air in the mediastinum described as a crunching or crackling sound over the heart synchronous with each contraction has, since his description of it, been known as Hamman's sign.

About 1930, and in subsequent years, the Macklins[19] by their laboratory investigations and voluminous writings greatly increased our knowledge of the pathogenesis of interstitial and mediastinal emphysema. They discussed the sequence of events and the factors involved in the rupture of the marginal alveoli and the transport of air by way of the sheaths of the pulmonary blood vessels to the hilus and thence to the mediastinum; furthermore, they stressed the potential dangers of this aberrent air within the thorax and employed the word "malignant" in describing[19c] these serious cases. The appropriate term "air block" was coined by one[19c] of them to describe the condition arising from interference of air in the lung, mediastinum and extensions therefrom with the vital functions of respiration and circulation.

The drift toward conformity in explaining the mechanism of mediastinal emphysema noted in the Nineteenth Century was halted in 1882 by a scientific inquiry reported by Champneys.[20a] This experimental investigation was conducted in the true Hunterian tradition, and the conclusions derived from it marked the beginning of a new concept of the manner in which air may reach the mediastinum. The experiments were performed on the bodies of still-born children that were subjected to artificial respiration after inserting a cannula into the trachea following a tracheotomy. Mediastinal emphysema occurred in seven out of 21 experiments, and it appeared that during inspiration the air travelled through the cervical incision into the tissues of the neck and mediastinum. He concluded that the conditions favoring the production of mediastinal emphysema were division of the deep cervical fascia during violent inspiratory efforts resulting from obstruction in the air passages, and that the route selected by the air was the space behind the deep fascia. A second report[20b] in 1884 contributed positive post-mortem findings of mediastinal emphysema in 16 of 28 fatal cases of tracheotomy, presumably all in young children.

Few records of mediastinal emphysema following tracheotomy are available in the literature prior to Champney's publication. Martini's equivocal cases[21] of two infants in 1858 who also had mediastinal abscess, Wilks' three cases[22] (two children and one adult) in 1860 and Bartel's boy[23] of seven in 1866, probably comprise the earliest recorded cases. All these patients died and were examined post-mortem, but no convincing theory of the course of the pathological changes was adduced.

It seems worthy of note that mediastinal emphysema as a complication of tracheotomy was either unrecognized or disregarded for a century or more. Although the operation is credited[24] to Asclepiades in 124 B. C., and was described by many surgical writers of antiquity, the first successful tracheotomy is ascribed to Brasovola[25] in 1546. The procedure was not often performed until advocated by Louis[26] for the removal of foreign bodies in the air passages in his celebrated "Second Memoir on Bronchotomy", published in 1759. Louis noted one fatal case of mediastinal emphysema occurring in a seven-year-old girl with a bean lodged in the trachea, but made no mention of its occurrence following tracheotomy. Similarly in this country Gross,[27] who popularized opening the trachea for the removal of foreign bodies, appeared to overlook the complication. Among accidents of tracheotomy, he stated that emphysema occasionally attended or followed the operation, but that it was more alarming than dangerous. His subsequent description was obviously that of subcutaneous emphysema of the neck. Weist[28] in 1882 reported on a study of 1000 cases of foreign bodies in the air passages to determine the propriety of bronchotomy in such accidents. The operation was performed in 338 cases with a mortality of 27.4 per cent. Among the 93 fatalities the causes of death were enumerated with no mention of mediastinal emphysema.

During this period one might suppose that mediastinal emphysema complicating tracheotomy for the relief of acute inflammatory obstruction in the upper air passages would occur with more frequency and be recognized. Likewise, the paucity of such reports is impressive. Bretonneau[29] in 1826 advocated and practiced tracheotomy for laryngeal obstruction in membranous croup which he regarded in most cases as diphtheria. Neither he nor his pupil Trousseau[30a] discussed the complication in their earlier manuscripts; however, in 1861 after extensive experience with the operation, Trousseau[30b] described the occurrence of emphysema of the neck and the resulting respiratory embarrassment when it invaded the chest. He emphasized the complication as being promoted by the rapid (stab) method of tracheotomy which he deprecated. In his opinion the more deliberate procedure was preferable, since with it there was less danger of the accident's happening.

Various reasons may be postulated for the apparent failure of these able clinicians to observe the complication which presumably arose occasionally. Many of the patients were moribund when the operation was performed, and died of their disease immediately, or too soon for emphysema to develop. Hemorrhage and failure to relieve asphyxia accounted for most of the operative deaths. Some cases may have been obscured by infection and by the so-called pneumonia, which was often recorded. Certainly a major cause was the inadequate delineation of the diagnostic features of air in the mediastinum, and the fact that the Roentgen ray had not yet been discovered.

Mediastinal Emphysema Complicating Tracheotomy.

The publication of Champney's article was soon succeeded by reports of other cases of post-tracheotomy mediastinal emphysema. Ewart and Roderick[31] in 1899 recorded the case of a five-year-old girl, ill with diphtheria, who died three days after tracheotomy. Autopsy revealed mediastinal and subcutaneous emphysema, which the authors attributed to weakening and bursting of the walls of the pulmonary alveoli from their distension by coughing. They distinguished the two routes by which air may enter the mediastinum in tracheotomy cases, and designated them respectively the extrinsic or inspiratory, by way of the recently divided fascial planes; and the intrinsic or expiratory type caused by distension and rupture of the lung alveoli under stress of bronchiolar obstruction. The latter method they considered to be less common but more serious on account of its supposed uncontrollability.

Leiner[32] in 1903 reported nine cases of mediastinal emphysema following tracheotomy in children. He believed that the air entered the neck wound and extended downward. He gave as reasons for this, the rapid appearance of mediastinal emphysema after the operation, the normal findings in the chest before operation, and the absence at autopsy of interstitial or subpleural emphysema in the lungs of the four patients who died. In 1939 Graebner[33] reported five children tracheotomized for acute laryngeal obstruction, in one of whom the mediastinal emphysema preceded the operation. In the same year, Michels[34] presented six instances of children with either mediastinal emphysema or pneumothorax developing after tracheotomy. Both Graebner and Michels attributed the complication to Macklin's theory. Neffson[35] took the contrary view after a careful discussion of the possible mechanisms in a report published in 1943. This was after an experience with 17 cases of pneumothorax and 30 of mediastinal emphysema in 126 tracheotomies in a 10-year period for the relief of acute non-diphtheritic obstructive infections of the larynx, trachea, and bronchi. Similar conclusions, namely that air is sucked by negative intrathoracic pressure through the tracheotomy wound along the deep cervical fascial planes into the mediastinum, were evinced in two other contemporary series of cases.

Goldberg and associates[36] reported 18 tracheotomies for croup with 12 mediastinal emphysemas and eight pneumothoraces. Their observations were complemented by experimental work on human cadavers. Forbes and associates[37] studied 120 children tracheotomized for acute laryngotracheobronchitis with an occurrence of postoperative mediastinal emphysema in 25 per cent and pneumothorax in 10 per cent of the patients. They also experimented with dogs and confirmed their beliefs.

During this same period Macklin was conducting his animal experiments and proving to his satisfaction the validity of his intrinsic theory of the mechanism by which air gains access to the mediastinum. His exhaustive treatise[19c] of 1944 detailed the applicability of his theory in explaining the development of mediastinal emphysema in many diverse circumstances unaccountable in any other way. A compelling example pertinent to the subject under discussion is the appearance of mediastinal emphysema before tracheotomy or without tracheotomy in occasional cases of acute laryngeal obstruction. Macklin argued that pulmonary interstitial emphysema was already present from alveolar distension and rupture caused by the respiratory obstruction, and that mediastinal emphysema and possibly pneumothorax resulted from its extension, thereby making the tracheotomy a co-incidental and not a causal element.

The route by which air reaches the mediastinum in the presence of any open neck wound when there is also obstruction to the airway continues to be a subject of much debate. The arguments for both theories are fully discussed in excellent reviews published in the decade from 1940 to 1950 by Goldberg,[36] Neffson,[35,38] Forbes[37] and Macklin.[19c] The truth probably is that both mechanisms operate in different cases and both may be involved together in certain individual instances.

For sake of completeness a third possible route to the mediastinum is mentioned, namely, by trauma to the apical pleura in performing a low tracheotomy. This was suggested by Wiethe[39] but is probably rare, as the injury has seldom been reported. As demonstrated by Macklin[19c] the resultant pneumothorax could only with great difficulty, if at all, rupture medially into the mediastinum to produce a pneumonomediastinum.

The incidence of mediastinal emphysema complicating tracheotomy is influenced directly and definitely by several well-recognized factors:

1. *The Urgency of the Operation as Measured by the Severity of the Dyspnea and Other Obstructive Signs.* The powerful inspiratory efforts greatly diminish the intrathoracic pressure and favor the indrawing of air through the open incision in the neck. If an interstitial emphysema has already started in the connective tissue of the lung it may continue to progress even though the obstruction is relieved. Once its need becomes apparent, tracheotomy should be performed as early as possible and not as a last desperate expedient.

2. *The Technique Employed and the Difficulty Experienced in Performing the Operation.* Champneys[24] considered the dangerous period to be the interval between division of the deep fascia and the introduction of the tube. Undue delay in this period increases the danger. Widespread dissection in the neck tissues and lifting the pretracheal fascia should be avoided. The incision should not be lower than necessary to avoid entering the superior mediastinum. Suturing of the skin close to the cannula was considered dangerous by Jackson[6] but was discounted by Neffson.[34]

firmed "the presence of an airway—or a small bronchoscope in the trachea during tracheotomy makes the operation an unhurried, tranquil procedure. It is probably the greatest single factor in lowering the mortality rate of tracheotomy *per se*, and in avoiding mediastinal emphysema and pneumothorax". Yet a few notes of warning concerning the procedure have been recorded. MacCready[12] felt that the additional instrumentation sometimes increased the respiratory efforts with its potential danger of inciting the complication. Wiley and Sugarbaker[13] found introduction of the tube impractical in the presence of many obstructive lesions. Holinger[14] drew attention to the risk of too low a tracheotomy while the head is hyperextended with a bronchoscope in place. When the bronchoscope is withdrawn and the head returns to its normal position the tracheal incision may be within the thorax. Subsequent coughing might then force air into the mediastinum.

3. The Adequacy of the Postoperative Supervision. This revolves mainly on the problem of maintaining constantly a clear airway. Repeated aspiration must be employed, if necessary, to eliminate obstruction in the lower passages. The correct placement of the tube within the lumen of the trachea at all times is of paramount importance. The necessity of close watching for a misplaced or blocked tube was emphasized by Reading[15] "that a patient is wearing a tracheotomy tube does not necessarily imply that he is able to breathe through it, nor does the fact that some air can be felt percolating from the wound in the region of the tube mean that he is breathing through the tube".

4. The Age of the Patient. Children develop the complication much more readily than older individuals. Many explanations have been advanced for this. Children tend to experience more acute degrees of respiratory obstruction than adults. They also present more technical problems to the surgeon. The operative field is smaller, with consequently a relatively longer incision. The greater looseness of the areolar tissue in the region facilitates separation of fascial layers and sometimes even allows the tube to be pushed down in front of the trachea in a struggling uncooperative child.

5. The Clinical Condition Necessitating Tracheotomy. Acute laryngotracheobronchitis deserves special consideration because this disease accounts for the great majority of tracheotomies, especially in children; and the incidence of a complicating mediastinal emphysema is much higher when the operation is required in its presence than in any other circumstance. Two reasons are given for this: *First,* the disease involves the *whole respiratory tract combining an acute upper respiratory* obstruction in the larynx with impediment in the lower passages by accumulation of thick secretions; dyspnea may persist *from the latter after the former is relieved by the tracheotomy. Second,* the extreme dyspnea and diffuse bronchopulmonary pathological changes tend to promote the production of pulmonary interstitial emphysema, according to the theory of Macklin. The double hazard of upper and lower respiratory obstruction inherent in acute laryngotracheobronchitis was discerned by Neffson and Wishik[46] as early as 1934, and is now generally acknowledged; in fact, the occasional failure of tracheotomy to afford satisfactory relief of dyspnea and the relatively high incidence of supervening emphysema and pneumothorax in this rather ill-defined disease-complex, has resulted in differing opinions as to the advisability of the operation in some cases. Although in favor of the operation when performed, if possible, as an elective procedure, Briggs and Heseltine[47] recently examined divergent points of view regarding this question.

Series of tracheotomies in patients with acute laryngotracheobronchitis as reported by Goldberg,[36] Neffson,[35] and Forbes,[37] with their high incidence of mediastinal emphysema, are in marked contrast to series where the operation was performed for other less complex obstructive conditions. Figi[48] in reporting 206 consecutive tracheotomies at the Mayo Clinic did not mention the occurrence of either mediastinal emphysema or pneumothorax. Only two of these patients had laryngotracheitis. Davis and associates,[49] in 155 operations, observed mediastinal emphysema as a complication in eight patients and pneumothorax in eight patients among 22 of the total having acute laryngotracheobronchitis. They expressed *surprise at the infreqeuncy of the complications in the series* as a whole, a fact they attributed to lack of routine postoperative chest roentgenograms. Fitzhugh (1941)[50a] in 100 tracheotomies which included 10 for acute laryngotracheobronchitis, reported two with pneumothorax, one of which also had recognized mediastinal emphysema. In a later series (1954)[50b] of 150 cases which apparently included none of acute laryngotracheobronchitis, there were three pneumothoraces. Subcutaneous emphysema and mediastinal emphysema were mentioned as often present but of a degree to cause no concern. Evans and Smalldon,[51] in New York Hospital, analyzed 50 cases of mediastinal emphysema from various causes, and found 10 occurred in tracheotomized patients, of whom five had acute laryngotracheobronchitis, and all of these were in patients three years of age or younger.

In recent years tracheotomy has been used successfully in an increasing number of entirely different conditions associated with obstruction by secretions in the lower respiratory tract. The purpose is to facilitate removal of obstructive secretions from the tracheo-bronchial tree and thereby prevent pulmonary complications and anoxia. The idea gained prominence through Galloway's work[52] on bulbar poliomyelitis in 1943, and Priest's subsequent studies[53] of the same disease. The new concept enormously increases the indications for the operation. These now include neurological disorders, severe debility, comatose states, botulism, drug poisoning, tetanus, severe head and chest injuries, and lengthy head and neck operations. Recent studies of the basic physiopathology involved, and reviews of these newer indications, with author references, have been made by Von Leden,[54] Fitzhugh and Morgan[50b] and Taylor.[55] Careful perusal of the original articles of the numerous authors cited revealed the enlightening fact that when tracheotomy was performed more or less as a prophylactic measure and not in the presence of acute high obstruction, the probability of mediastinal emphysema was rarely mentioned as a complication, and then usually as only an inconsequential possibility.

During the 12-year period from 1943 to 1954, inclusive, there were 232 tracheotomies performed on 225 patients in St. Luke's and Roosevelt Hospitals in New York City. The accompanying table classifies the indications for the operation according to the degree of respiratory difficulty. Not all the acute conditions represent emergency procedures with little regard to sterile technique, but all the patients in this category had dyspnea and other signs of definite obstruction at the time of operation This study is in no sense a statistical one dealing with averages or medians, but rather one concerned primarily with the unexpected and the unpredictable Yet it is deserving of notice that all of the eight instances of mediastinal emphysema occurred in the urgent group, and in three of these the patient had acute laryngotracheobronchitis

MEDIASTINAL EMPHYSEMA COMPLICATING TRACHEOTOMY

	Adults	Children under 12 years of age	Total
Acute laryngotracheo bronchitis	5 (1 M E)	14 (2 M E)	19
Other acute conditions	53 (2 M E)	16 (2 M E)	69
Malignant neoplasms of larynx			
Acute conditions	21 (1 M E)		21
Elective operations	78		78
Other elective operations	42	3	45
	199	33	232

In this series the number of patients operated upon for acute laryngotracheobronchitis seems disproportionately small They reflected the total number of admissions for this disease, and are possibly explained by the fact that many of them are confused with diphtheria before admission and are taken directly to hospitals for contagious diseases Among the elective operations there was none for poliomyelitis, as these cases, during the period reviewed, were being handled in other institutions *equipped with special isolation facilities*

Physiopathology

Mediastinal emphysema may remain confined within the mediastinum, or it may escape into the deep and subcutaneous tissues of the neck, into one or both pleural cavities, or through the diaphragmatic openings into the retroperitoneal space The amount of air invading the mediastinum determines the pressure that is exerted. Hamman[18c] emphasized that when sufficient pressure develops, air always escapes from the mediastinum. Escape into the neck and contiguous subcutaneous regions is easily detected and is usually considered evidence of mediastinal emphysema. This inference is on sound premises when emphysema of the mediastinum is the sequel of an initial pulmonary interstitial emphysema. In such cases, as for example, spontaneous mediastinal emphysema and that occurring during expiratory straining against a closed glottis, there is justification in terming the presence of subcutaneous emphysema in the neck as pathognomonic of mediastinal emphysema. The finding is not necessarily diagnostic in wounds of the chest communicating with the lung, or where air enters the fascial planes from the buccal or nasal cavities; likewise, air may leak around a tracheotomy opening or through a fracture site in the larynx and cause subcutaneous emphysema in the neck without the inevitable co-existence of mediastinal emphysema. Notwithstanding, in these latter instances one might profitably heed the admonition

> *"Look to it well and say you are well warn'd."*
>
> —Shakespeare: Henry VI, Part 1, Act II, Sc. 4, 1. 103.

After subcutaneous emphysema the next most frequently diagnosed sequel of mediastinal emphysema is pneumothorax. This represents escape of air under pressure through the thin pleural surfaces of the mediastinum into the pleural cavity. This actuality was clearly demonstrated experimentally by Joannides and Tsoulos[56] in dogs, and verified by Keis,[57] and Barrie,[55] and Goldberg[30] in human cadavers. In living patients the critical mediastinal pressure necessary to rupture into the pleural cavity remains to be determined. Goldberg's experiments[30] seemed to indicate that it must be above 20 mm. of mercury. Keiss and Barrie both believed that sufficiently high pressure could be attained to accomplish this in the presence of dyspnea from respiratory obstruction, even though an open neck wound was present. They found no reason to question the belief that air may be sucked through an open wound into the mediastinum and thence enter the pleural cavities. Seidl,[59] on the other hand, from his experiments on dogs and clinical observations on thyroidectomy patients, could not conceive of sufficient pressure being built up in the mediastinum by this method to cause its rupture into the pleural cavity If air entered the mediastinum by this route, which he conceded as possible, he claimed that it should be absorbed or escape through the original neck wound or through any drainage opening Seed[60] concurred, and used the findings to further his argument in favor of Macklin's theory to explain mediastinal emphysema and pneumothorax in thyroidectomy cases

The importance of these rival theories is emphasized anew in the presence of a complicating tension pneumothorax If the original source of air were an ipsilateral pulmonary interstitial emphysema from ruptured alveoli, further leakage might be lessened or arrested with compression of the lung by the pneumothorax This action would be non-operative if the air were entering the mediastinum through the fascial planes of the neck The prognostic significance of the primary route of entry of air is obvious

One more aspect of the interrelationship of mediastinal emphysema and pneumothorax should be noticed The rupture of a subpleural emphysematous bleb on the surface of the lung is often offered as an explanation for spontaneous pneumothorax A valvular mechanism is invoked to explain positive pressure in the pleural cavity It seems most improbable for mediastinal or subcutaneous emphysema to result from pneumothorax produced in this way Much higher pressures are required to rupture the normal visceral pleura than the thin mediastinal pleura Air passes rather easily from the mediastinum into the pleural cavity, but almost never in the reverse direction, as demonstrated by experimental and clinical observations [19e, 18b] If pulmonary interstitial emphysema is present, it is theoretically possible for air to proceed peripherally toward the surface of the lung,[61] but Macklin[19b] proved that pressure gradients took most of it along the blood vessel sheaths toward the hilus If air entered by way of the neck the mediastinal emphysema must perforce have preceded the pneumothorax

The third escape route for air from the mediastinum is through the diaphragm about the aorta and esophagus to the pressure that is exerted. Hamman[18c] emphasized that when sufficient pressure develops, air always escapes from the mediastinum. Escape into the neck and contiguous subcutaneous regions is easily detected and is usually considered evidence of mediastinal emphysema. This inference is on sound premises when emphysema of the mediastinum is the sequel of an initial pulmonary interstitial emphysema. In such cases, as for example, spontaneous mediastinal emphysema and that occurring during expiratory straining against a closed glottis, there is justification in terming the presence of subcutaneous emphysema in the neck as pathognomonic of mediastinal emphysema. The finding is not necessarily diagnostic in wounds of the chest communicating with the lung, or where air enters the fascial planes from the buccal or nasal cavities; likewise, air may leak around a tracheotomy opening or through a fracture site in the larynx and cause subcutaneous emphysema in the neck without the inevitable co-existence of mediastinal emphysema. Notwithstanding, in these latter instances one might profitably heed the admonition

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> —Shakespeare: Henry VI, Part 1, Act II, Sc. 4, 1. 103.

After subcutaneous emphysema the next most frequently diagnosed sequel of mediastinal emphysema is pneumothorax. This represents escape of air under pressure through the thin pleural surfaces of the mediastinum into the pleural cavity. This actuality was clearly demonstrated experimentally by Joannides and Tsoulos[56] in dogs, and verified by Keis,[57] and Barrie,[58] and Goldberg[36] in human cadavers. In living patients the critical mediastinal pressure necessary to rupture into the pleural cavity remains to be determined. Goldberg's experiments[36] seemed to indicate that it must be above 20 mm. of mercury. Keiss and Barrie both believed that sufficiently high pressure could be attained to accomplish this in the presence of dyspnea from respiratory obstruction, even though an open neck wound was present. They found no reason to question the belief that air may be sucked through an open wound into the mediastinum and thence enter the pleural cavities. Seidl,[59] on the other hand, from his experiments on dogs and clinical observations on thyroidectomy patients, could not conceive of sufficient pressure being built up in the mediastinum by this method to cause its rupture into the pleural cavity. If air entered the mediastinum by this route, which he conceded as possible, he claimed that it should be absorbed or escape through the original neck wound or through any drainage opening. Seed[60] concurred, and used the findings to further his argument in favor of Macklin's theory to explain mediastinal emphysema and pneumothorax in thyroidectomy cases.

The importance of these rival theories is emphasized anew in the presence of a complicating tension pneumothorax. If the original source of air were an ipsilateral pulmonary interstitial emphysema from ruptured alveoli, further leakage might be lessened or arrested with compression of the lung by the pneumothorax. This action would be non-operative if the air were entering the mediastinum through the fascial planes of the neck. The prognostic significance of the primary route of entry of air is obvious.

One more aspect of the interrelationship of mediastinal emphysema and pneumothorax should be noticed. The rupture of a subpleural emphysematous bleb on the surface of the lung is often offered as an explanation for spontaneous pneumothorax. A valvular mechanism is invoked to explain positive pressure in the pleural cavity. It seems most improbable for mediastinal or subcutaneous emphysema to result from pneumothorax produced in this way. Much higher pressures are required to rupture the normal visceral pleura than the thin mediastinal pleura. Air passes rather easily from the mediastinum into the pleural cavity, but almost never in the reverse direction, as demonstrated by experimental and clinical observations.[19e,18b] If pulmonary interstitial emphysema is present, it is theoretically possible for air to proceed peripherally toward the surface of the lung,[61] but Macklin[19b] proved that pressure gradients took most of it along the blood vessel sheaths toward the hilus. If air entered by way of the neck the mediastinal emphysema must perforce have preceded the pneumothorax.

The third escape route for air from the mediastinum is through the diaphragm about the aorta and esophagus to the retroperitoneal tissues. This pathway is utilized less frequently than the superior aperture into the neck. Sweet[62] explained this by the fact that air in body spaces tends to rise in contradistinction to liquids which gravitate downward. He speculated that this physical law applied in mediastinal emphysema when the patient was in the usual semi-recumbent position, and that it accounted for the more common appearance of emphysema in the subcutaneous tissues of the neck.

A small amount of air in the mediastinum may produce only trivial symptoms, or none at all; whereas large amounts may cause grave disturbances and may even lead to death. Jehn and Nissen[63] proved by animal experiments that no recognizable symptoms appeared until the normally negative intramediastinal pressure reached about zero. If more air was introduced signs of cardiac tamponade, falling blood pressure with dyspnea and cyanosis soon supervened. These changes progressed rapidly depending on the amount of pressure and the rapidity of its increase. The effects were the result of increased mediastinal pressure causing mechanical interference to the circulation by compression of the large veins entering both sides of the heart. This action was demonstrated experimentally in rabbits by Ballon and Francis[64] and confirmed clinically by Jessup.[65] That the pernicious consequences of increased mediastinal pressure as noted by these investigators was due to the rapidity and the uncontrolled extent of its formation is illustrated by comparison with the benign reactions observed during the induction of orderly diagnostic artificial pneumomediastinum.

The diagnostic injection of 40 to 150 cc. of air into the mediastinum of each of 40 children by Castellanos and Pereiras[66] caused no ill effects. In adults, Bariéty and associates[67] routinely injected 400 to 700 cc. of air in 20 minutes, creating a final pressure of 8 to 15 cm. of water with only insignificant symptoms. They attributed this remarkable tolerance to avoidance of sudden and massive distension of the mediastinal cellular tissue with resultant flooding of the escape avenues. The availability and spontaneous use of these escape channels thus constitutes a powerful factor in regulating abnormal intramediastinal pressures.

Torrey and Grosh[68] noted the dramatic relief of symptoms and prompt improvement in the patient's condition as high intrathoracic pressure was relieved by the appearance of subcutaneous emphysema. Conversely, it has been often commented that when there are alarming signs of increased mediastinal pressure there is usually little subcutaneous emphysema. A reciprocal relationship between mediastinal pressure and subcutaneous emphysema was noted experimentally by Ballon and Francis.[64] The release of air under pressure from the mediastinum into the pleural cavity does not offer such a safe escape. The capacity of the pleural cavity is limited, and dyspnea and cyanosis are augmented rather than relieved if tension pneumothorax supervenes. If the condition is bilateral, life itself is in grave peril. In his monograph on acute laryngotracheobronchitis, Neffson[38] estimated the mortality to be less than 20 per cent in unilateral pneumothorax, but about 90 per cent in bilateral cases.

Much has been learned about the anatomy of the region since the time of Ambroise Paré and the succeeding Seventeenth Century. The mediastinum is no longer believed to be the firm rigid structure described by Crooke[69] in 1616, whose action is to support and steady the heart and to separate it from the lungs, and the lungs from each other, so that all may function independently. Increased knowledge of the physiology of the mediastinum was made possible by the introduction of differential pressure anesthesia in 1904 by Sauerbruch.[70] A means was thus provided for maintaining respiration when the chest had been laid open, and one or both lungs were exposed to the atmospheric pressure, thereby permitting operations to be performed on thoracic viscera. Surgeons could then verify clinically Graham's modern concept[71] of a yielding and mobile mediastinum and contents; yet much is still unknown regarding the physio-pathology of the mediastinum. Bariéty[67] has epitomized it well by stating that one does not deal here with a well-defined closed space to which accurate physical laws may be applied, but rather with a passage open upwards and downwards, whose size, capacity, structure, suppleness and permeability are variable from one subject to another. These variables must be taken into account when evaluating an individual case of mediastinal emphysema.

Symptoms and Physical Signs.

Once one is familiar with the syndrome of mediastinal emphysema and its sequelae its identity should not be difficult. The diagnosis depends primarily upon the surgeon's awareness of the possibility. Symptoms usually appear during the operation or within a few hours of its termination. The clinical signs have been described[72,73,74] more comprehensively in discussions concerning spontaneous emphysema of the mediastinum in which the picture is clearly defined and the symptoms are not mingled with those of an operation or an antecedent medical condition. Schwartz, McIlroy and Warren[75] observed the essential similarity in the clinical picture of all cases of mediastinal emphysema, however caused, but noted that any fatal cases have invariably occurred in the secondary type.

Pain in the chest is the most constant symptom and is characteristically precordial or substernal. It may be severe with anginal type of radiation simulating organic heart disease.[76] This has been noted particularly in spontaneous cases but according to Scott[77] it is a feature in any serious increase in mediastinal pressure from whatever cause. Pain of this sort and severity necessitates differentiation from the two diseases most commonly confused with the entity: acute pericarditis, and acute coronary occlusion with myocardial infarction. In mediastinal emphysema the pain typically alters with changes of position of the body, varies with respiration, and may be aggravated by swallowing or turning the head. Surgeons are aware of the significance of this kind of pain when it occurs in chest injuries. In these traumatic cases Gatellier[78] emphasized the importance of searching for a developing mediastinal emphysema at the onset of diaphragmatic pain, especially when associated with unexplained dyspnea.

Dyspnea in some degree is nearly always present, and as indicated by Jessup[65] is directly related to the extent of embarrassment of vital structures in the mediastinum by trapped air. When very severe and accompanied by cyanosis, pneumothorax should be suspected. That mediastinal emphysema alone may be responsible, however, must be borne in mind. The experience of Torrey and Grosh[68] showed that severe dyspnea and cyanosis materially lessened when air escaped into the subcutaneous tissues of the neck. Gumbiner and Cutler[79] established relief of these symptoms in newborn children by removing air from the mediastinum by a needle.

No constitutional signs usually accompany the syndrome except in severe cases. Draper[73] said that this fact provided negative evidence of inestimable value in distinguishing mediastinal emphysema from more serious cardio-vascular disorders. There is no associated fever, leucocytosis, or increased sedimentation rate. Until the condition is severe and, therefore, otherwise obvious, there is no change in the pulse rate or blood pressure. Electrocardiographic changes may occur[72,76] but they are inconstant, transient, and not characteristic.

The most distinctive physical sign of mediastinal emphysema is mediastinal crepitation (Hamman's sign). This consists of crackling, bubbling or crunching sounds heard over the heart with each contraction. These may be heard only during systole, but sometimes they are present throughout the whole cardiac cycle with accentuation during systole. They may vary greatly during the episode and with change of position or phase of respiration. The sounds are typical and do not occur with any other condition. When present the sign is pathognomonic. Its absence, however, does not rule out emphysema of the mediastinum. The air must have migrated to the anterior mediastinum to be heard. In Macklin's experiments[19b] air tended to accumulate first in the middle and posterior mediastinal spaces. The sign usually persists for six to eight days, but may last only a few hours and thereby be missed. It is less constantly observed in children than in adults. Goldberg[36] found Hamman's sign in only six of 31 children with proven mediastinal emphysema.

Diminished or obliterated cardiac dullness, sometimes replaced by hyperresonance, is less frequently reported, but its presence has diagnostic value.

Subcutaneous emphysema has long been acknowledged as a common and reliable sign of mediastinal emphysema. It is easily recognized and is often the first intimation that air is contained within the mediastinum. It usually appears first in the neck, thence spreading over the face and thorax, and may finally become generalized over most of the body. In the absence of an open chest injury or a penetrating wound of the upper air passages, it must be considered presumptive evidence of mediastinal emphysema. Subcutaneous emphysema is the palpable sign of a mechanism by which intramediastinal pressure is relieved. Its extent, therefore, is no accurate gauge of current conditions in the mediastinum. The important matter is the quantity of air still retained in the mediastinum.

Pneumothorax, as a sequel to mediastinal emphysema, must by definition, follow it; but when pneumothorax does develop, it is discovered simultaneously in the majority of cases. Occurring with operations in the neck, mediastinal emphysema and pneumothorax probably have an identical origin, and because of their frequent conjunction they are usually considered as parts of the same entity. The dramatic signs of pneumothorax, often sudden and accompanied by shock, overshadow the lesser ones of a preceding mediastinal emphysema. During an operation or in the post-operative period while the patient may be still under the influence of anesthesia, the onset of pneumothorax may provide the first and only evidence of air under pressure in the mediastinum. The gravity implicit in the diagnosis should not be underestimated.

Roentgenographic demonstration of air in the mediastinum is pathognomonic of emphysema. Anterior-posterior views show widening of the mediastinal shadow with irregular dark streaks parallel to the cardiac border on either side. Small amounts of air, unless localized, may not be visualized on this view. Aisner and Franco[74] directed attention to the error of eliminating mediastinal emphysema on the basis of a single film taken in the postero-anterior projection and advised serial films if necessary. Like Gumbiner and Cutler[79] they stressed the value of lateral projections in revealing air behind or in front of the heart. Evans and Smalldon[51] considered lateral and oblique views essential before one could rule out mediastinal emphysema. Seed[60] was of the same opinion but pointed out the difficulty of obtaining a picture in a dyspneic bed-patient by means of a portable X-ray machine. Schinz, Boensch, Friedl and Uehlinger,[80] in their recent textbook of Roentgen diagnosis, recommended fluoroscopy of the mediastinum in the *oblique position. The awkwardness of using this technique* with an ill postoperative patient is a disadvantage.

The importance of routine Roentgenograms of the chest to detect mediastinal emphysema and pneumothorax after operations on the neck cannot be over-emphasized. Some of the patients with these complications present few or no symptoms, and the *condition would otherwise remain unrecognized.* Davis[13] believed numbers of cases are missed in this manner. Forbes[37] advised examination by fluoroscopy or by Roentgenogram within six hours in all tracheotomy cases. In his series the incidence increased from 25 per cent to 41 per cent in the case of mediastinal emphysema and from 10 per cent to 15 per cent in that of pneumothorax when the patients were checked by Roentgen examination.

REPORT OF TRACHEOTOMY CASES

Case 1 E P, 1954. Acute Laryngotracheobronchitis This 10 year old white boy had fever, cough, and increasing dyspnea for three days On admission his temperature was 103° F, pulse 140 and respirations 50 and irregular Dyspnea was severe with cyanosis and chest retraction Roentgenographic examination of the chest immediately before tracheotomy was negative Rapid improvement in symptoms occurred after the operation On the following day marked subcutaneous emphysema was present in the neck and anterior chest wall Hamman's sign was positive, and a Roentgenogram revealed air in the mediastinum but none in the pleural cavities The child was given oxygen, steam inhalations, and antibiotics The air disappeared from the mediastinum in three days and from the subcutaneous tissues in five days The tracheotomy tube was removed on the sixth day, and the patient was discharged from the hospital as cured on the tenth day.

Case 2: M. W, 1953. Acute Laryngotracheobronchitis A 21-year old white woman had acute respiratory symptoms for 36 hours Her temperature was 103° F, pulse 100, and respirations 22 on admission Marked inspiratory stridor and extreme dyspnea were present Preoperative Roentgenogram of the chest was negative. Tracheotomy afforded relief of the dyspnea Subcutaneous emphysema in the neck and upper part of chest appeared the next day. Positive signs of mediastinal emphysema were verified by Roentgenographic examination No pneumothorax occurred. The patient was treated with antibiotics. The tube was removed in six days. At that time examination of the chest was negative, but there was still some subcutaneous emphysema in the neck and external chest wall

Comment: Two cases of mediastinal emphysema following tracheotomy for acute laryngotracheobronchitis are presented: one in a child and one in an adult. Both were of moderate degree and both exhibited subcutaneous emphysema without pneumothorax.

Case 3: A. M., 1943. Acute Laryngotracheobronchitis. A 2-year-old white boy was acutely ill with fever and dyspnea. He had been in the hospital a year previously for a similar but milder attack, and had been treated only medically. This time the child had been ill for one week and on admission was extremely dyspneic and in poor condition. Roentgenograms of the chest showed slight patchy consolidation in both lungs and a small pneumothorax at the right base. No mediastinal emphysema was noted, but a lateral view was not taken. Tracheotomy was performed with a bronchoscope in place in the trachea. Dyspnea was only moderately relieved by the operation and was not improved by tracheal suction. The patient became cyanotic, and died suddenly one-and-a-half hours after the operation. Permission for autopsy was not granted, but the clinical signs on physical examination signified a bilateral tension pneumothorax. No subcutaneous emphysema was present.

Comment: This is a case of acute fulminating laryngotracheobronchitis with evidence of pneumothorax before tracheotomy, suggesting the presence of pulmonary interstitial emphysema according to Macklin's theory. It illustrates the potential seriousness of mediastinal emphysema when the pleural cavity is utilized as the escape route for the aberrent air under pressure.

Case 4: J. L., 1943. Impacted Foreign Body in the Larynx. This 3-year-old white boy claimed he had swallowed a nut which a stranger had given him just before he was brought to the hospital. Distinct wheezing and some dyspnea were present. Breath sounds were diminished on the right side. Roentgengraphic examination did not demonstrate a foreign body but did reveal a slight obstructive emphysema of the entire right lung. Without anesthesia a 5 mm. bronchoscope was readily introduced, and a smooth hard-shelled nut was found obstructing the orifice of the right main bronchus. Difficulty was experienced in grasping the foreign body satisfactorily. During withdrawal it was dislodged from the forceps in the subglottic region. Severe laryngeal spasm immediately ensued, and the patient's airway was completely obstructed. Emergency tracheotomy was performed and normal respirations were resumed. During the operation and before the lumen of the trachea was entered the child made several powerful inspiratory efforts. The foreign body remained so tightly wedged between the cords that it could not be readily extracted. The decision was made to leave it in place above the cannula for a while rather than to administer a general anesthetic to facilitate its removal. The lower air passages and lungs were clear. Several hours later, during the night, slight subcutaneous emphysema in the suprasternal notch was noted, but no other abnormal signs were present. Oxygen was given, although no dyspnea was observed. Sixteen hours after the operation severe dyspnea and cyanosis suddenly supervened. Bilateral tension pneumothorax was diagnosed and both pleural cavities were aspired with needles and syringes, but the patient went into shock and died. Autopsy was performed under water five hours after death. Subcutaneous emphysema was present in the neck and right supraclavicular fossa. The mediastinum contained multiple blebs of air distending its entire connective tissue structure. Both lungs were collapsed by tension pneumothorax. All pleural surfaces were smooth, showing no emphysematous blebs and no signs of injury. The pleurae revealed no adhesions. There was no evidence of trauma to the parietal pleura or to either lung at the apex. The foreign body proved to be a pistachio nut in its shell measuring 14x8x7 mm. in size. It fell free into the trachea during the post mortem examination. There was no indication of laceration or other injury of the bronchi or of the trachea below the tracheotomy site. The microscopic examination of the lungs revealed almost complete atelectasis of both lungs. Some alveoli were distended, and a few showed rupture of their walls. Careful search, however, failed to disclose any air in the interstitial tissues of the lungs or in the perivascular or peribronchial sheaths.

Comment: Direct aspiration of air through the open neck wound during the operation was thought to be the cause of the mediastinal emphysema in this case. Why the pressure continued to build up by this route in the face of a subsequently clear airway is not readily explained. Pulmonary interstitial emphysema would satisfactorily reconcile the sequence of events, but no confirmatory evidence could be elicited by postmortem examination which was conducted under optimum conditions.

Case 5: J. G., 1951. Carcinoma of Larynx with Edema. Hoarseness for nine months and dysphagia for two weeks had not impelled this 63-year-old white laboring man to seek medical advice, but dyspnea for 24 hours did. Acute edema of the larynx superimposed on a large carcinoma necessitated an emergency tracheotomy. During the procedure the patient was severely dyspneic and had extreme retraction of the chest. Several sutures were inserted to close a rather long incision. One hour after the operation subcutaneous emphysema appeared in his neck and chest wall. Increasing dyspnea pointed toward mediastinal involvement. Prompt removal of the sutures was accompanied by escape of air and relief of symptoms. Signs of both mediastinal emphysema and subcutaneous emphysema were gone in six days.

Comment: The evidence in this case suggests that the emphysema was caused by too tight closure of the incision around the tracheotomy tube. Jackson[40] has warned about this danger.

Case 6: J. M., 1953. Acute Edema of Larynx Superimposed on Postradiation Fibrosis. An emergency tracheotomy was performed on this 46-year-old white man immediately after admission to the hospital. This was made necessary by an acute edema of the larynx with obstruction causing stridor, dyspnea and some cyanosis of a few hours' duration. History obtained later revealed that he had had a tracheotomy performed in another hospital 10 years earlier for carcinoma of the larynx. The operation had been followed by prolonged treatment with radium and the patient had worn the tube for seven years. No symptoms except chronic hoarseness for the last three years were noticed until the day of his present admission.

Tracheotomy was extremely difficult because of the excessive degree of dense fibrosis in the tissues of the region. This obviously was a sequel to the previous operation and subsequent radiation therapy. Considerable bleeding was encountered which further delayed a troublesome dissection. The following day subcutaneous emphysema was evident in the neck and right side of the chest. The patient complained of considerable pain in the substernal region, but had no elevation of temperature or leucocytosis. Roentgenographic examination revealed a pneumothorax at the left base and a possible mediastinal emphysema (no lateral view taken). Symptoms improved with disappearance of signs of abnormal air in 12 days. The patient left the hospital with the tracheotomy tube in place. Direct laryngoscopy, afterward, revealed distortion and partial cicatricial stenosis of the larynx, but no evidence of regrowth of neoplastic tissue.

Comment: Widespread dissection of the neck tissues and delay in entering the trachea in the presence of acute laryngeal obstruction predispose to the development of mediastinal emphysema in tracheotomy cases. When such technique is unavoidable prudence calls for increased vigilance during the postoperative period.

Case 7: R. H., 1954. Revision of Tracheotomy. When two months old, this Negro boy had a herniorrhaphy performed for an incarcerated congenital left inguinal hernia. While reacting from the anesthesia (open inhalation method) be regurgitated and aspirated some gastric contents. Bronchial aspiration by catheter and also through a 3½ mm. bronchoscope failed to relieve the dyspnea and cyanosis, so an emergency tracheotomy was done, using a No. 1 tube. The acute condition was alleviated. Subsequent attempts to decannulate the trachea were unsuccessful because of chronic subglottic edema causing obstruction above the tracheotomy opening. This was attributed to too high placement of the tube in the trachea. One month after the primary operation, the tracheotomy wound was revised and the cannula inserted considerably lower in the airway. The operation was performed under general ether anesthesia administered by mask over the open tracheal wound and under no pressure. No difficulty was encountered with the anesthesia. Immediately after completion of the operation extensive subcutaneous emphysema appeared in the neck and upper part of the chest, and the patient became slightly dyspneic. Roentgenograms showed extensive emphysema of the mediastinum and subcutaneous tissues of the neck and chest wall, with clear lungs and no pneumothorax. Preoperative films had been negative. It required nine days for the air beneath the skin and in the mediastinum to disappear clinically and as shown on Roentgen films.

Comment: The mediastinal emphysema in this case was probably caused by the incision and dissection being carried too low in the neck, thereby opening the fascial planes close to the superior mediastinum. Pulmonary interstitial emphysema associated with the anesthesia cannot be ruled out, but no positive pressure was used and no dyspnea was observed during the operation.

Case 8: J. S., 1954. Resection of Trachea for Stenosis caused by Tracheomalacia. Two months before admission this 60-year-old white woman had had a thyroidectomy performed in another hospital. A large substernal gland diagnosed as Hashimoto's struma had been excised under general anesthesia without tracheal intubation. The immediate postoperative period was stormy with much dyspnea and cyanosis. The dyspnea had persisted in severe degree and was accompanied by loud expiratory wheezing, characteristic of tracheal obstruction.[81] Her voice was good, and vocal cord function was normal. Bronchoscopy revealed a smooth concentric stenosis of the trachea at the level of the fifth ring This region dilated during inspiration but during expiration collapsed to a caliber which would barely permit passage of an ordinary bronchoscopic suction tube Routine Roentgenograms of the chest were negative Laminographs showed the trachea to be in the midline with a smooth tapering tracheal stenosis opposite the second and third thoracic vertebrae and in addition, showed a poorly defined bilateral soft tissue density in the substernal region

Under local anesthesia the original transverse neck incision was reopened and the trachea exposed The second and third rings were cut for the insertion of a rubber breathing catheter extending past the site of the stenosis Much dense fibrous scar tissue made exposure and freeing of the trachea very difficult The fifth tracheal cartilage had been destroyed and was replaced by a band of fibrous tissue which constricted the lumen to ½ cm in diameter This area for a distance of ¾ cm was resected and the upper and lower segments of the trachea were approximated with interrupted sutures of No 32 stainless steel wire The anastomosis appeared to be air tight The tracheal lumen could now accommodate the No 5 tracheotomy tube which was put in place through a new opening in the skin above the orginal incision The primary wound was closed and two small Penrose drains were inserted Biopsy of the residual thyroid tissue later confirmed the diagnosis of Hashimoto's type of chronic thyroiditis

Subcutaneous emphysema appeared in the neck on the day following operation Roentgenographic examination revealed slight emphysema in the mediastinum and in the fascial planes of the neck This disappeared in five days Other symptoms were absent No pneumothorax occurred The tracheotomy tube was removed in ten days Subsequent bronchoscopy demonstrated a normal tracheal lumen The patient has remained well

Comment This is an example of mediastinal emphysema occurring after a difficult neck dissection and tracheotomy in the presence of respiratory obstruction The case might logically be included among those occurring during thyroidectomy It was considered improbable that air was blown directly through the site of the tracheal resection because the dyspnea had been completely and permanently relieved before the anastomosis was accomplished.

Mediastinal Emphysema Complicating Thyroidectomy

In 1946 Meek[82] remarked that for mediastinal emphysema to follow surgical procedures on the neck was a logical conclusion if one considered both the relationship of the cervical fascial spaces to the mediastinum and the mechanics of breathing. The first report of mediastinal emphysema complicating a neck operation other than tracheotomy was by Buford[83] in 1918 The procedure was a thyroidectomy and excision of tuberculous nodes of the neck In 1924 Gold[84] reported four cases following thyroidectomy In 1934 Kers[57] collected seven cases in the literature; all followed thyroidectomy; all had associated pneumothorax; all were fatal. In subsequent years there have appeared sporadic reports of other cases, nearly always accompanied with pneumothorax and nearly always extremely serious. The low incidence and extreme gravity of these reported cases has led to the suspicion that mediastinal emphysema complicating neck operations has occurred much more frequently than the literature would indicate and that the complication often might be mild and unrecognized.

Next to tracheotomy, thyroidectomy has accounted for most of the cases of mediastinal emphysema occurring as an operative complication. Operations on the neck, other than thyroidectomy, however, have been reported as the responsible agent. The factors involved are analogous. The case reported by Schmidt[55] in 1931 followed a jugular vein ligation; that by Ackerman and Bricker[56] in 1941 followed a radical dissection of cervical lymph nodes, and one of the two by Herrmann[57] in 1942 followed resection of the upper segment of the esophagus for carcinoma. Additional cases in the literature to 1949 have been listed by Seed.[60] Since that year two significant series of cases have appeared. Bowden and Schweizer[58] in 1950 reported 18 cases of pneumothorax and two of mediastinal emphysema arising during operative procedures about the head and neck in a five-year period at Memorial Hospital, New York. Aiken and Smith,[59] in England in 1953, found either mediastinal emphysema or pneumothorax as a complication in eight patients among 900 block dissections of the neck. An increased incidence of the complication or an increased awareness of its development, or both, must be inferred.

The theory of the mechanism by which mediastinal emphysema develops in thyroidectomy cases, as in tracheotomy cases, has been the subject of much debate. The majority of the earlier investigators favored the theory that air entered the neck wound and was sucked into the mediastinum along the fascial planes by negative pressure. Barrie[58] had three patients who died of mediastinal emphysema and pneumothorax following thyroidectomy. The autopsy findings and his subsequent experiments on cadavers convinced him that air entered by way of the wound. Keis[57] had previously performed similar experiments and reached the same conclusion. Ackerman and

Bricker[86] were strongly of the same opinion. Like Keis they found no post-mortem evidence of pulmonary interstitial emphysema in their patient. Aiken and Smith,[89] after discussing the possible routes of entry, also concluded that air entered by way of the neck, but they restricted the pathway to the carotid sheath. They stressed the sucking sound heard at operation as being indicative of this mechanism. Barrie[38] had also described this sound and thought that it signified air being drawn into the tissue spaces.

The important role of respiratory obstruction in the development of mediastinal emphysema during neck operations was early appreciated. Schmidt,[85] and Jessup[65] both stressed the danger of inspiratory dyspnea from tracheal compression. As long ago as 1924 Gold[84] suggested having a tracheoscope in place during the operation to overcome upper tracheal obstruction. Pressure by a goiter is not the only cause of dyspnea in thyroid operations. Edema, hemorrhage into the wound,[90] excessive mucus, surgical interference with the recurrent laryngeal nerves,[45] laryngeal spasm,[91] tracheal collapse,[48] and intubation difficulties[85] have all been blamed. For significant amounts of air to enter the mediastinum directly along the fascial planes from an open neck wound seems to require the simultaneous existence of some degree of respiratory obstruction.

Inextricably connected with the issue of mediastinal emphysema in thyroid surgery is the element of anesthesia. These operations are customarily performed under general anesthesia with the agent administered sometimes by the inhalation method but commonly through an intratracheal tube and under positive pressure. Coughing and signs of tracheal and bronchial obstruction are familiar incidents during these procedures. *The tendency for interstitial emphysema of the lung* to develop under these conditions is well established. The onset of emphysema of the mediastinum is a logical sequence. In more than half the cases of mediastinal emphysema during thyroidectomy analyzed by him, Seed[60] noted difficulty in breathing either as a result of removing an intrathoracic goiter or from trouble with the anesthesia. Seidl[59] emphatically attributed mediastinal emphysema in thyroidectomy patients to pneumothorax; all were fatal. In subsequent years there have appeared sporadic reports of other cases, nearly always accompanied with pneumothorax and nearly always extremely serious. The low incidence and extreme gravity of these reported cases has led to the suspicion that mediastinal emphysema complicating neck operations has occurred much more frequently than the literature would indicate and that the complication often might be mild and unrecognized.

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Bricker[56] were strongly of the same opinion Like Kers they found no post mortem evidence of pulmonary interstitial emphysema in their patient Aiken and Smith,[89] after discussing the possible routes of entry, also concluded that air entered by way of the neck but they restricted the pathway to the carotid sheath They stressed the sucking sound heard at operation as being indicative of this mechanism Barrie[58] had also described this sound and thought that it signified air being drawn into the tissue spaces

The important role of respiratory obstruction in the development of mediastinal emphysema during neck operations was early appreciated Schmidt[8] and Jessup[65] both stressed the danger of inspiratory dyspnea from tracheal compression As long ago as 1924 Gold[34] suggested having a tracheoscope in place during the operation to overcome upper tracheal obstruction Pressure by a goiter is not the only cause of dyspnea in thyroid operations Edema hemorrhage into the wound,[90] excessive mucus surgical interference with the recurrent laryngeal nerves[4] laryngeal spasm tracheal collapse,[48] and intubation difficulties[8] have all been blamed For significant amounts of air to enter the mediastinum directly along the fascial planes from an open neck wound seems to require the simultaneous existence of some degree of respiratory obstruction

Inextricably connected with the issue of mediastinal emphysema in thyroid surgery is the element of anesthesia These operations are customarily performed under general anesthesia with the agent administered sometimes by the inhalation method but commonly through an intratracheal tube and under positive pressure Coughing and signs of tracheal and bronchial obstruction are familiar incidents during these procedures The tendency for interstitial emphysema of the lung to develop under these conditions is well established The onset of emphysema of the mediastinum is a logical sequence In more than half the cases of mediastinal emphysema during thyroidectomy analyzed by him, Seed[60] noted difficulty in breathing either as a result of removing an intrathoracic goiter or from trouble with the anesthesia Seidl[59] emphatically attributed mediastinal emphysema in thyroidectomy patients obstruction of respiration during the operation. He believed the increased pressure caused alveolar distension and rupture leading to pulmonary interstitial emphysema.

The intrapulmonary pressure developed during intratracheal insufflation anesthesia may be the determining factor. Too high a pressure of gas may be entering the lungs. In his experiments on cats and dogs, Macklin[19a] conclusively demonstrated the effects of high intrabronchial pressure. Air could break through the overdistended alveoli into the interstitial tissue of the lung and thence track along the perivascular channels to the hilus and rupture into the mediastinum. That surgical emphysema can develop in this way from anesthesia alone was forcefully illustrated in Bradshaw's patient.[92] Symptoms of the condition, including a small pneumothorax, appeared after intubation and before any operation was attempted. As Macklin[19c] said, some cases of pulmonary interstitial emphysema and pneumomediastinum attributed to the surgical procedure in reality arise during the administration of the anesthesia. Marcotte, Phillips, Adams and Livingstone[93] confirmed that intrabronchial pressure of 24 mm. Hg. could produce mediastinal emphysema in dogs. Such a rise might be caused by coughing or by any obstruction to the outflow of gas. Important to the subject under discussion is Marcotte's conclusion that intrabronchial pressure depended chiefly upon the patency of the outlet rather than upon the pressure at which the gases were forced into the lungs, and the observation that once emphysema had developed, it might be increased at a pressure lower than that necessary to initiate it. Spence[94] considered the administration of an anesthesic at a pressure of 20 mm. Hg. as unsafe. He advised a pressure of 10 mm. Hg. or less when using an intubation technique.

Injury to the mucosa of the upper air passages is often alluded to as a possible route of entry for air into the mediastinum. Such an injury might be a laceration occurring during the insertion of an intratracheal tube. Air then could be blown directly into the mediastinum and rapidly distend it. Aiken and Smith[89] believed that the accident occasionally happened and they cited examples from the literature. A feature was the rapid development of subcutaneous emphysema after induction of anesthesia Most writers on the subject have dismissed this course as unlikely and have not noted such injuries [60, 86] Even if a mucosal laceration were present, Macklin[19c] advised careful search for interstitial emphysema of the lungs as the more probable source of mediastinal air Despite the potential hazards of intratracheal anesthesia, Meek[8] thought the free airway which the tube afforded was good prophylaxis against the production of mediastinal emphysema

The fundamental problem in thyroidectomy is essentially the same as that in tracheotomy An open neck wound exists in the presence of an inadequate airway Many tracheotomy patients are children with their greater susceptibility for developing mediastinal emphysema and in whom the degree of preoperative dyspnea is often alarming Thyroidectomy, on the contrary, is performed chiefly in adults, but the operation is more prolonged, and it is subject to many intercurrent factors tending to promote respiratory obstruction during the procedure The occurrence of mediastinal emphysema during thyroidectomy and comparable neck operations has generated discussion paralleling that occasioned by the same complication in tracheotomy Conflicting theories of the mechanism of production of mediastinal emphysema during these operations have been expounded in several recent articles

Baillie,[8] Keis,[57] Ackerman and Bricker,[86] and Aiken and Smith[89] all concluded that the air entered the mediastinal tissues by way of the neck wound They found no evidence of injury to the mucosa of the upper respiratory tract that would permit direct entrance of air into the mediastinum Neither did they find any injury to the apical pleura Pulmonary interstitial emphysema was eliminated by consistently negative findings at autopsy

Seed[6] ably discussed the evidence in favor of Macklin's theory He quoted the observations and experimental findings of Seidl[90] who was also convinced that this mechanism was responsible for the complication Although aspiration of air through the open wound was the most frequent explanation given as the cause of mediastinal emphysema and pneumothorax following thyroidectomy, Seed found that it could not satisfactorily explain all cases, moreover, the failure to demonstrate pulmonary interstitial emphysema and mediastinal emphysema at autopsy in all patients believed to have had these conditions had been reconciled by Macklin[10c]. He had emphasized that the diagnosis might easily be missed unless a special underwater technique was employed. He had stressed, further, that if the examination were delayed a number of hours, the air in the interstitial tissues of the lung and in the mediastinum would diffuse and not be discovered. Concerning Macklin's theory, Seed asserted "this theory will satisfactorily explain the history and findings of all the reported cases of mediastinal emphysema and pneumothorax following thyroidectomy. No other one will."

Bowden and Schweizer[33] presented an excellent summary of the anatomical considerations relating to these complications in neck surgery. By opening the middle layer of the deep cervical fascia which envelops the thyroid gland, a free passageway into the mediastinum is provided. Between the middle and deep layers of the deep fascia in the midline space containing the trachea and esophagus there is a direct passage into the mediastinum. After analyzing their 20 cases they thought that in two patients mediastinal emphysema and and pneumothorax followed rupture of the marginal alveoli causing pulmonary interstitial emphysema; but in the majority of cases they were convinced that the air entered the superior mediastinum through the operative site during exaggerated respiratory effort.

The diagnosis of mediastinal emphysema occurring alone can rarely, if ever, be made during the course of a neck operation. The classical signs and symptoms cannot be elicited, or are masked by the anesthesia. Even in the postoperative period the isolated complication is seldom reported as a diagnosis. The condition is usually identified by the appearance of air in one of the escape channels. This circumstance brings conviction that many cases of mediastinal emphysema of mild degree have escaped recognition. Subcutaneous emphysema in the neck may occur during or immediately following the operation and is presumptive evidence of mediastinal emphysema. Tension pneumothorax primarily creates the symptoms which direct attention to the lungs and mediastinum. A characteristic sign[60,88] of evolving pneumothorax is some variation in the patient's normal respiratory pattern during the operation. In the presence of positive tension within the pleural cavity there is typically a relatively long expiratory phase alternating with a very short inspiratory phase. Severe dyspnea and cyanosis may supervene. At the onset of such signs the surgeon should suspect the presence of mediastinal emphysema and pneumo thorax.

REPORT OF THYROIDECTOMY CASES.

During the 12 year period from 1943 to 1954, inclusive, 1651 thyroidectomies were performed in St. Luke's and Roosevelt Hospitals in New York City. Mediastinal emphysema occurred as an operative complication in three of these cases.

Case 9 W W Operation January 14, 1947. Subtotal thyroidectomy was performed on this 56 year old white man for removal of a diffuse toxic goiter Preoperative medication of propylthiouracil for six weeks and Lugol's solution for one week had lowered his basal metabolic rate from plus 53 per cent to plus 24 per cent. The operation was done under general inhalation anesthesia using an endotracheal tube Operating time was 90 minutes No difficulty was experienced with the anesthesia or the operation. A small Penrose drain was inserted. Three hours after completion of the operation, the patient had severe pain in the chest, mostly in the substernal region and on the right side Dyspnea, cyanosis and tachycardia rapidly supervened. Physical examination revealed loud crepitations in the precordial region during systole (Hamman's sign) and signs of tension pneumothorax on the right side with extreme mediastinal shift to the left. Subcutaneous emphysema was present in the neck, anterior chest wall, right axilla and right posterior chest wall Roentgenographic examination confirmed the diagnosis of mediastinal emphysema and right pneumothorax with total collapse of the lung. Immediate underwater drainage of the right pleural cavity was instituted and was continued for two days. The patient improved rapidly. Hamman's sign persisted for three days On the sixth postoperative day a Roentgenogram showed almost complete expansion of the right lung. Slight subcutaneous emphysema was still present seven days after the operation. On the tenth day the patient was out of bed and without symptoms

Comment: This case is somewhat unusual in that no difficulty was encountered with the operation or with the anesthesia. It illustrates the fact that the complication is unpredictable and that its occurrence cannot always be anticipated. The efficacy of prompt and adequate decompression for the relief of tension pneumothorax is demonstrated.

Case 10 M H Operation October 20, 1950. This 41-year old white man, a mechanic, had a subtotal thyroidectomy for a diffuse toxic goiter that had caused symptoms for one-and a-half years. Administration of propylonstrate pulmonary interstitial emphysema and mediastinal emphysema at autopsy in all patients believed to have had these conditions had been reconciled by Macklin[19c]. He had emphasized that the diagnosis might easily be missed unless a special underwater technique was employed. He had stressed, further, that if the examination were delayed a number of hours, the air in the interstitial tissues of the lung and in the mediastinum would diffuse and not be discovered. Concerning Macklin's theory, Seed asserted "this theory will satisfactorily explain the history and findings of all the reported cases of mediastinal emphysema and pneumothorax following thyroidectomy. No other one will."

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Case 10 M. H. Operation October 20, 1950. This 41-year old white man, a mechanic, had a subtotal thyroidectomy for a diffuse toxic goiter that had caused symptoms for one-and a-half years. Administration of propylthiouracil for 14 months had lowered his basal metabolic rate from plus 64 per cent to plus 4 per cent. Lugol's solution was given for one week before the operation. A very large goiter required two hours for its removal. Intravenous pentothal and closed gas-oxygen-ether inhalation anesthesia was employed without tracheal intubation. During the second half of the operation irregular respirations for 30 minutes were noted and attributed to light anesthesia. The skin incision was closed without drainage.

A few hours after the operation the patient had slight precordial pain, but no other symptoms except traces of subcutaneous emphysema in the neck. The pain persisted until the third postoperative day, when it became severe and was accompanied by moderate dyspnea and increased pulse rate (90 per minute). No cyanosis was observed. The pain shifted to the substernal region and did not radiate. It varied with respiration and with body position, being worse on deep inspiration and being relieved by a sitting posture. It was made worse by swallowing. These symptoms lasted four more days and then disappeared. The patient was well and able to leave the hospital on the eighth postoperative day. During the period of his acute pain, his temperature and blood pressure remained normal. Blood count and sedimentation rate were normal. Physical examination of the heart and lungs revealed no abnormalities. No relief was obtained by the administration of nitroglycerine. An electrocardiogram obtained on the fourth postoperative day showed R. T. segment changes suggestive of pericardial disease. A subsequent tracing three days later showed changes consistent with subsiding pericardial involvement On the basis of these findings the medical consultant ruled out coronary or myocardial disease. A provisional diagnosis of pleuropericarditis secondary to mediastinal emphysema or to a low-grade mediastinitis was made. In the absence of any constitutional signs of inflammation and with the prompt subsidence of symptoms the presence of mediastinal emphysema was considered more likely. Unfortunately, although Roentgenographic examination was suggested, no films were obtained.

Comment: The diagnosis of mediastinal emphysema was not proved absolutely in this case but the clinical findings, both positive and negative, cannot be explained by any other condition. The clinical picture is reminiscent of that described[72,75] in some cases of spontaneous mediastinal emphysema simulating cardiac disease.

Case 11: I. B. Operation December 15, 1952. Eighteen years previously, this 39-year-old white woman had had a partial thyroidectomy for exophthalmic goiter. She now had a recurrent non-toxic nodular goiter with suspected malignant changes. Total removal of a large hard right lobe was accomplished by operation under general inhalation anesthesia with an endotracheal tube in place. Intubation was difficult. The operation required two-and-one-quarter hours and during the latter half the patient had a severe spell of coughing lasting 10 minutes. A rubber tissue drain was left in place after the skin incision was closed. Precordial pain began a few hours after completion of the operation. It was moderately severe, did not radiate, and was increased by movement and by coughing and swallowing. Crepitations synchronous with the heart beat (Hamman's sign) were audible Signs of pneumothorax were present on the right side. A Roentgenogram demonstrated 20 per cent collapse of the right lung but did not show air in the mediastinum. (No lateral view was taken). Very little dyspnea was noted. The patient had a low-grade fever for six days, with no leucocytosis. She was given penicillin for three days, and had sedatives for pain. Hamman's sign remained positive for eight days. Roentgenographic examination on the seventh postoperative day showed moderate re-expansion of the right lung and a very evident mediastinal emphysema. All symptoms had disappeared by the tenth postoperative day when the patient left the hospital. There was never any subcutaneous emphysema. The pathological diagnosis of the excised thyroid gland was Riedel's struma with plasma cell infiltration.

Comment: A positive Roentgenogram is diagnostic of mediastinal emphysema, but a single negative report does not necessarily rule out its presence. It is submitted that many rather typical cases similar to this one may occur and are not reported. Milder degrees of the condition with little or no pain or with insufficient pressure to produce pneumothorax may often be unrecognized or disregarded.

Treatment.

A small amount of air in the mediastinum requires no active treatment but is a signal for vigilance. The air is usually absorbed in a few days and has no influence on the prognosis. Larger quantities of air necessitate measures for controlling the source and for providing an outlet for the air already accumulated. Diligent efforts to eliminate any respiratory obstruction must be instituted. For dyspnea or cyanosis inhalations of oxygen should be administered, but never under positive pressure if pulmonary interstitial emphysema from ruptured alveoli is a possibility. Artificial respiration is contraindicated. Both Keis[57] and Ackerman[86] mentioned that this *tends to increase the mediastinal pressure and facilitate the* production of pneumothorax. Pain may be alleviated by analgesics, and excessive coughing should be controlled. Emphysema in itself and unaccompanied by infection does not require antibiotics.

Signs of increasing mediastinal pressure are an indication for aspiration of air from the mediastinum. Incision through the suprasternal notch down to the trachea to permit escape of air through the superior aperture is commonly recommended. In tracheotomy and thyroidectomy cases, however, a recent neck wound is already present. It would seem logical to open the wound widely and even to insert a rubber catheter into the mediastinum if necessary. Barrie,[58] on the contrary, oval of all drains and closure of the neck wound to her entry of air by aspiration. This action would ted and even contraindicated if the emphysema pulmonary interstitial emphysema. Suction ap- incision at the base of the neck by the method of onsidered by many as unnecessarily complex and value. Aspiration of the mediastinum[79] can be ost directly by the insertion of a needle attached in the second or third interspace near the sternal pointed toward the midline.

pneumothorax is present immediate decompres- ssary. This may be accomplished by simple needle f the pleural cavity. If repeated aspirations fail eaccumulation of air continuous underwater drain- e employed.

eous emphysema, even if extensive, is not in itself n for other than conservative treatment in medias- sema. In fact those patients who display subcu- hysema are, as a rule, less seriously ill than those . Multiple skin incisions may facilitate the disap- air from the subcutaneous tissues.

SUMMARY.

al emphysema occurring as a complication of on the neck is discussed. Eight cases complicating y and three cases following thyroidectomy are pre-

The factors in the two operations tending to promote the condition appear to be analogous. An open neck wound exists in the presence of some degree of respiratory obstruction. The increased respiratory effort due to the inadequate airway causes exaggeration of the normally negative intramediastinal pressure and at the same time tends to increase the positive intrabronchial and intra-alveolar pressure.

Experimental evidence and clinical observation, as revealed in the literature, indicate that two different mechanisms operate in these cases to produce mediastinal emphysema. Air may enter the neck wound and be drawn along the fascial planes into the mediastinum by negative intrathoracic pressur air under increased pressure may rupture overdistended monary alveoli causing an interstitial emphysema of the l with subsequent migration of the air along the perivascu sheaths to the hilus. Either mechanism, or possibly both t gether may be the responsible agent in an individual case.

Mediastinal emphysema complicating tracheotomy and other neck operations probably occurs more frequently than a review [illegible] would indicate. An active awareness of the possibility by the surgeon may be lacking or many cases of a mild type may be disregarded as of little consequence. Minimal degrees of mediastinal emphysema often may be unrecognized because no symptoms attributable to the condition exist. Routine postoperative Roentgenograms taken with the correct technique may [illegible] the decisive diagnostic measure.

The history of the subject suggests that the danger of mediastinal emphysema occurring with these operations may never be absent. Since the cause cannot be completely eradicated the effects may be controlled or even surmounted by watchfulness and, when necessary, by prompt and appropriate intervention.

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A VARIATION IN TECHNIQUE OF FACIAL NERVE DECOMPRESSION.[*]

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New Orleans, La.

INTRODUCTION.

Decompression of the facial nerve in its course through the temporal bone from the geniculate ganglion to the stylomastoid foramen is essential in the treatment of facial paralysis of otitic origin. Decompression of the vertical portion of the nerve from the horizontal semicircular canal to the stylomastoid foramen has been accepted by many as an adjunct in the treatment of Bell's palsy and has recently been advocated by Williams and associates[1] in the treatment of hemifacial spasm.

It was my belief that if facial nerve decompression and neurolysis could be performed with little discomfort, short hospitalization and postoperative convalescence, no change in hearing, and no other complications incident to the operation, it could be advised more freely to patients with Bell's palsy or hemifacial spasm. A technique was, therefore, devised by which the facial nerve could be decompressed from the stylomastoid foramen to or near the geniculate ganglion, without interruption of the ossicular chain, with preservation of hearing and with minimal trauma to the nerve and its blood supply.

REVIEW OF THE LITERATURE.

Historical: In 1812 Charles Bell[2] clearly showed that the VIIth cranial nerve is responsible for the motor innervation of the facial muscles and that it is not responsible for the pain of tic douloureux as was previously believed. In 1822 he[3] published a study on the anatomy and philosophy of

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expressions, separating voluntary and emotional responses of the face. Since his time peripheral facial paralysis not due to injury has been commonly known as Bell's palsy.

In 1879 Drobnik[4] united the peripheral end of the VIIth nerve to the central end of the external branch of the XIth nerve in a patient with facial paralysis with partial return of function. Numerous methods of anastomosis between the distal end of the VIIth nerve and the central end of various adjacent cranial nerves have since been suggested; in 1895 Ballance[5] anastomosed the VIIth to the XIth; in 1903, Korte[6] the VIIth to the XIIth, and in 1926, the VIIth to the IXth was reported in two patients by Watson-Williams[7]. Often a straight face in repose and some voluntary movement resulted, but these procedures were generally unsatisfactory *because of associated movements and complete lack of emotional* response of the facial musculature.

Love and Cannon[8] reviewed the cases of anastomosis of the VIIth to the XIth cranial nerves at the Mayo Clinic performed from 1926 to 1949. Most of the cases resulted from removal of VIIIth nerve neuromas and results of anastomosis in these were good in 75 per cent of the cases, but lack of emotional response and associated movements still existed.

Alt[9] was the first to advocate decompression in 1908 when he decompressed the facial nerve above and below an erosion in the facial canal produced by a cholesteatoma with recovery of the facial paralysis. Fourteen years later Ney[10] suggested repair within the temporal bone and in 1930 Bunnell[11] used a graft from the sural nerve to bridge a gap in the facial nerve in the temporal bone; but it is to the work of Ballance and Duel[12] set forth in their monograph that we owe our current concepts of intratemporal facial nerve surgery.

Anatomy—Localization of the lesion in facial paralysis is of utmost importance in any contemplated therapy. Theoretically, the lesion can be accurately localized; practically the lesion cannot be localized so specifically. The motor nucleus of the VIIth nerve lies deep in the lower portion of the pons; supranuclear pathways consist in *1.* crossed pyramidal tract, *2.* uncrossed pyramidal tracts (supplying only the temporal branch fibers), *3.* extrapyramidal tracts from the emotional center in the thalamus, and *4.* connection with the Vth, IInd and VIIIth cranial nerves; thus, supranuclear or central facial paralysis involves only voluntary control of the lower portion of the face, and voluntary control of the forehead and complete emotional response are retained.[13] The facial nerve emerges from the lower border of the pons at the cerebellopontine angle, enters the internal auditory meatus with the VIIIth cranial nerve and courses anterolaterally to the geniculate ganglion. A lesion in this area produces loss of function of the VIIIth nerve as well as peripheral facial paralysis. The greater superficial petrosal nerve leaves the facial nerve at the geniculate ganglion, and geniculate lesions theoretically are localized by decreased lacrimation, loss of taste on the anterior two-thirds of the homolateral side of the tongue, hyperacusis and peripheral facial paralysis. The facial nerve leaves the geniculate ganglion entering the tympanic cavity just posterosuperiorly to the cochleariform process; entering the facial canal, it courses posteriorly for 8 mm., this portion being called the tympanic portion, where at a point just below the horizontal semicircular canal it turns inferiorly, still within the facial canal from which it makes its exit at the stylomastoid foramen 7 to 8 mm. below. The almost right angle bend below the horizontal canal is called the pyramidal portion. The stapedius branch of the nerve is given off from this portion, and the chorda tympani 2 to 3 mm. lower from the mastoid portion; thus, the presence or absence of hyperacusis and taste of the anterior two-thirds of the homolateral side of the tongue aids in localization of the lesion in peripheral facial paralysis.[14]

The blood supply of the facial nerve is from the internal auditory artery, the superior petrosal branch of the middle meningeal artery and the stylomastoid branch of the posterior auricular artery. Anastomoses occur above the pyramidal portion of the nerve but not below,[15] which possibly explains return of function of the chordae tympani and stapedius branches with persistent facial paralysis. After its exit from the stylomastoid foramen, the nerve turns abruptly forward dividing into its various branches deep to the superficial lobe of the parotid gland; a lesion in this area (tumor, trauma) presents no problem in localization.

Etiology: Interruption of transmission of the neural impulses within the facial canal is generally conceded to be the mechanism responsible for Bell's palsy. Compression of the nerve without interruption of the axis cylinders does not cause failure of nerve conduction.[16] Hilger,[15] Kettel[17] and Sullivan[14] believed that the paralysis is due to local ischemia, which interrupts conduction and which may be accompanied by edema. This ischemia may be precipitated by exposure to cold, emotional factors and hereditary predisposition. Tickle[18] listed Bell's palsy as a toxic type of facial paralysis, and Collier[19] believed that many cases are viral in origin. There are a few patients who presumably have inadequate facial canals, as recurrent Bell's palsy is not infrequent.[17]

Pathology: The appearance of the facial nerve and the mastoid cells in patients with Bell's palsy who have undergone decompression was studied in 50 consecutive cases by Kettel.[20] Twenty per cent of the cases had necrosis of the mastoid cells, 36 per cent showed necrosis of the Fallopian canal, in two cases the nerve was exposed by necrosis, and in 56 per cent of the cases the nerve was grossly edematous in its vertical portion; microscopically there was no evidence of cellular infiltration. Cawthorne[21] reported constriction of the nerve at the stylomastoid foramen and bulging above in 12 of 14 patients who had undergone decompression.

Medical Treatment: Nonsurgical management of Bell's palsy has been directed toward relief of the ischemia and reestablishment of the blood supply by the early use of vasodilating drugs, sympathetic blocking drugs, and stellate ganglion block.[15,22] Recently, the use of adrenal cortical hormones in the early treatment of Bell's palsy has given encouraging results.[23] Support of the paralyzed muscles by the use of hooks, tape or intra-oral splints is generally advocated. Controversy exists concerning the use of galvanic or actual massage. Some writers [15,19] believe that the early use of galvanic massage retards fibrosis of the facial musculature whereas others[24] oppose galvanic stimulation because they believe that its use promotes fibrosis.

Selection of Cases of Bell's Palsy for Decompression: Bell's palsy may persist only a few days as a transient block or may progress to complete Wallerian degeneration. In approximately 80 per cent of patients with Bell's palsy spontaneous remission occurs.[11,24] The difficulty lies in determination of the 20 per cent who will need decompression. Once degeneration has occurred, recovery is never complete and is always marked by some mass movement and loss of function of the forehead muscles, whether recovery is spontaneous or follows decompression. Decompression of the facial nerve in Bell's palsy permits development of collateral circulation, reestablishment of circulation in the ischemic vessels and development of new vessels from the adjacent soft tissue.

Emphasis has been placed on loss of response to Faradic stimulation by Sullivan,[14] Cawthorne[21] and Tickle;[18] it is considered unreliable by Kettel[20] and Martin.[25] The presence of pain, completeness of the palsy, and rapidity of onset influence selection of cases for decompression. The advised time lag between onset of the paralysis and decompression varies from immediate decompression[14] to six months,[16] but averages eight to twelve weeks. All agree that the facial musculature must respond to galvanic stimulation if decompression is to be of value. Decompression is advocated enthusiastically by many[11,12,14,17,18,25] and with greater reluctance by others.[15,19] Use of electromyography may aid in detecting those patients who will recover spontaneously.[19,25]

Cryptogenic Hemifacial Spasm: This is an affection of the facial nerve and may be differentiated from facial tics (which are thought by some to be compulsion neuroses) by the following points: *1*. Hemifacial spasm involves only muscles innervated by the facial nerve; *2*. spasmodic facial tic is associated with tension relieved by performance and with anguish by suppression, and can be initiated and stopped at will whereas hemifacial spasm can not; *3*. hemifacial spasm occurs during sleep, but habit tics do not.[27] Cryptogenic hemifacial spasm must also be differentiated from hemifacial spasm secondary to other lesions of the facial nerve, as Bell's palsy and intracranial aneurysm.[28]

The etiology of cryptogenic hemifacial spasm is unknown.

Wartenberg[20] postulated that the facial nucleus contains two motor systems analogous to the pyramidal and lower motor neuron systems and that a lesion of the upper system produces hemifacial spasm and the lower Bell's palsy. Williams and associates[1] believed that hemifacial spasm is a lower motor neuron lesion, because a stroke does not affect the condition; hemifacial spasm is marked by weakness and contractures of the muscles, and known lesions of the lower motor neuron frequently produce hemifacial spasm. They found evidence of compression of the facial nerve in its vertical portion by fibrous tissue in four of eight patients with hemifacial spasm treated by decompression. Their immediate results were excellent, but the spasm recurred in two patients; permanent facial paralysis resulted in one, and there was a 50 per cent return of symptoms in another.

Patients with hemifacial spasm are miserable, embarrassed and handicapped. Heretofore treatment has been worthless, and there are few spontaneous remissions. If only partial or temporary relief can be obtained by decompression, it would seem worthwhile.

THE STUDY.

Decompression of the facial nerve by the postauricular route presents the disadvantages of difficulty in identification of the nerve at its exit from the stylomastoid foramen because of excessive bleeding, possible damage to the stylomastoid artery, postoperative discomfort and scarring, and an oblique view of the operative field. The usual endaural approach for decompression, in which the nerve is first identified just below the horizontal semicircular canal, or at a point just above the stylomastoid foramen by the blind removal of solid bone, presents the possibility of injury to the nerve before exposure is recognized.

The nerve is most readily identified in its tympanic course, where the Fallopian canal stands out in relief and adjacent landmarks are distinct; therefore, decompression without danger of trauma is best begun in this area, where the bony covering is quite thin. The nerve may be identified in this area by the following technique:

Technique: An open S-shaped endaural incision is made 2 to 3 mm. medial to the auricular cartilage beginning at seven o'clock in the right ear and five o'clock in the left ear. The incision is carried posteriorly and superiorly around the meatus through the periosteum to twelve o'clock and then extended superiorly superficial to the temporal fascia and muscle for a distance of 2 to 3 cm. The periosteum and superficial tissues are elevated from the mastoid cortex posteriorly, inferiorly and superiorly. A second incision is then made 1 to 2 mm. lateral to the first, sloping posteriorly beginning at the beginning of the first incision and extending to twelve o'clock, and forward 1 cm. so that a wedge-shaped

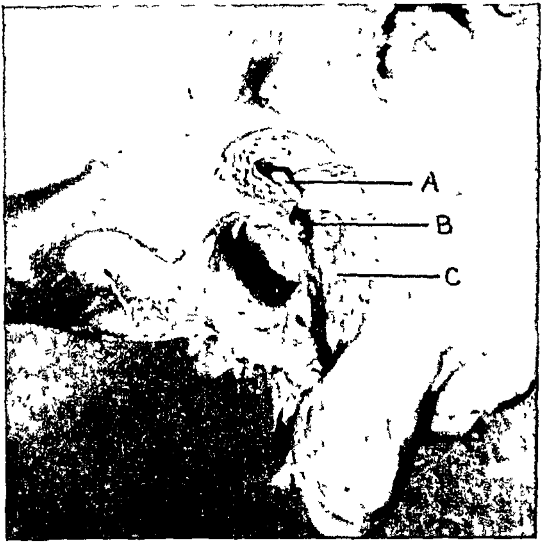

Fig 1 Photograph of temporal bone
(A) Short process of incus and horizontal semicular canal
(B) Surgically created opening into tympanic cavity
(C) Facial nerve

strip of skin, subcutaneous tissue and temporal fascia is removed (according to the technique of House[30]). A three-bladed endaural retractor is then inserted and the field exposed. Cortical bone is then removed with motor driven cutting burrs over the aditus, antrum and mastoid process, extending anteriorly until the antrum and short process of the incus are exposed and inferiorly and medially to the digastric ridge. The removal of bone is carried posteriorly to the sinus plate or less if the sinus lies far posteriorly. The superior and posterior osseous external auditory canal wall is then thinned with cutting burrs to expose the short process of the incus adequately; care is taken not to disturb the short process or the fossa incudis, and not to expose the incudòmalleolar joint. The osseous canal is not lowered except in the patient with a forward lying sinus, in which case it is lowered only as much as needed for adequate exposure. After the canal has been thinned to a thickness of 1 to 2 mm., a hole is drilled with a 1 mm. polishing burr 1 to 1.5 mm. inferior to the lowermost extension of the short process of the incus, at a depth just superficial to the horizontal semicircular canal, and in an anteromedial direction paralleling the osseous external auditory canal (see Fig. 1). This enters the tympanic cavity at the level of and lateral to the stapes. This opening is then enlarged circumferentially, leaving a post of bone 1 to 1.5 mm. in width between the short process of the incus and the opening. The Fallopian canal is readily visualized through this opening in its course between the horizontal ampulla and the stapes, and in a roomy temporal bone the nerve may be seen up to the cochleariform process and geniculate ganglion. Visualization of the nerve in this area establishes the depth and general direction of the nerve and serves to orient its position. Removal of the dense bone overlying the Fallopian canal is then begun with cutting burrs sweeping from above downward. The operator uses as guides to the position of the nerve, the Fallopian canal itself above, and the junction of the digastric ridge and the tympanomastoid suture line inferiorly, which marks the position of the stylomastoid foramen. Under direct illumination and magnification bone is removed from the posterior and lateral surfaces of the Fallopian canal, slowly and with frequent irrigation to cool the area, until a bluish translucency, much as is seen in the formation of the fenestra in the fenestration operation, is developed over the entire length of the nerve from the pyramidal portion above to the stylomastoid foramen below. The thin bone overlying the nerve is then carefully removed with the use of dental excavators and small curettes, and the stylomastoid foramen is enlarged posteriorly, care being taken not to injure the stylomastoid artery. The nerve is carefully inspected, and its sheath is slit along its posterior surface with a sharp myringotomy knife to avoid injury to the arteriolar trunk, which lies on the lateral surface of the nerve, as pointed out by Sullivan.[13]

The incision is then closed with interrupted 4-0 black silk sutures without drainage. The external auditory canal is packed tightly with selvage vaseline gauze strips, and a mastoid dressing is applied. Postoperatively, antibiotics are given routinely. The patient is discharged from the hospital on the third to fifth postoperative day and is seen in the office on the fifth postoperative day for removal of the sutures and the packing in the auditory canal. The dressing is left off.

This technique has been performed on five patients for Bell's palsy and three for cryptogenic hemifacial spasm. There have been no surgical injuries to the nerve incident to decompression, no postoperative complications, and no change in hearing in the operated ear. The period of absence from work has been minimal. The following cases are illustrative of results of decompression for Bell's palsy and for hemifacial spasm.

REPORT OF CASES.

Case 1. K. B., age 43 years, awoke with complete right facial paralysis on Nov. 15, 1952. This was accompanied by mild pain behind the ear. There was no history of chilling or other precipitating factors. When first seen on Jan. 18, 1953, complete medical, neurological, laboratory and Roentgenographic surveys revealed no abnormalities except complete right peripheral facial paralysis. On Jan. 23, 1953, approximately nine weeks from the onset of Bell's palsy, facial nerve decompression was done; the nerve appeared edematous throughout its vertical portion. The patient was discharged from the hospital on the fifth postoperative day. Examination three and one half months postoperatively showed no change in hearing, a normal ear on otoscopy, and 75 per cent normal function of the right facial musculature (see Fig. 2).

Case 2. L. A., a white man, 47 years of age, was seen on Jan. 11, 1955, because of severe spasmodic contractions of the muscles of the right side

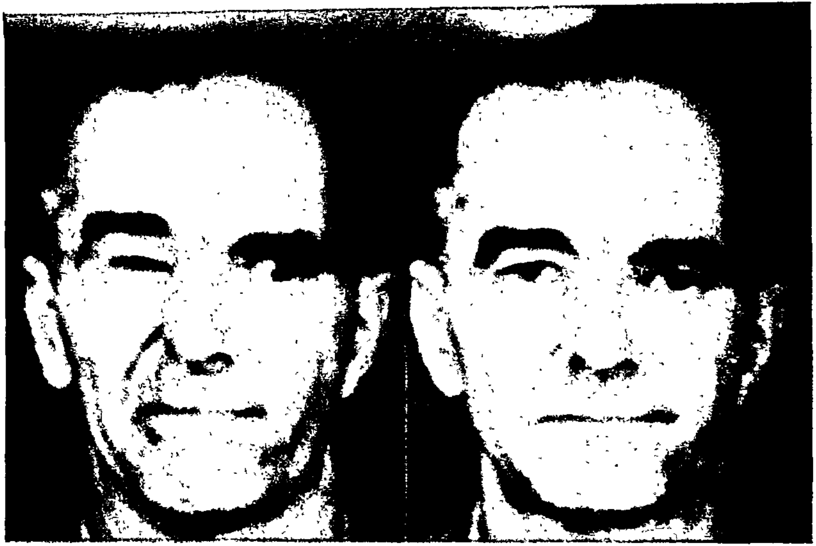

Fig. 8. (A) Photograph of patient with hemifacial spasm in spasm.
(B) Photograph of patient not in spasm.

of the face, especially severe about the eye. He had no control over this symptom, which had been present for six years, and had become more severe in the past four months. Thorough examination of the ears, nose and throat revealed no abnormalities. Examination by a neurosurgeon disclosed no neurologic abnormality. On Feb. 9, 1955, the right facial nerve was decompressed from just below the cochleariform process to the stylomastoid foramen, and neurolysis was performed. There was no gross abnormality of the nerve. The patient was discharged from the hospital on the fifth postoperative day. The immediate postoperative result was excellent; there was no facial weakness, and the spasm was completely relieved for four months; however, six months postoperatively the spasm returned to 50 per cent of its previous severity (see Fig. 3).

SUMMARY.

In eight patients with Bell's palsy or hemifacial spasm, a variation in technique of facial nerve decompression has given better results than established techniques used in the past. By this method the facial nerve is decompressed from the stylomastoid foramen to or near the geniculate ganglion, without interruption of the ossicular chain, with preservation of hearing and with minimal trauma to the nerve and its blood supply. Although this series is much too small to justify conclusions, the absence of complications, lack of effect on the hearing, and minimal hospitalization and morbidity indicate that it may be a useful adjunct and worthy of trial in these patients.

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REPAIR OF CHRONIC CENTRAL PERFORATIONS OF THE TYMPANIC MEMBRANE: BY REPEATED ACID CAUTERY; BY SKIN GRAFTING.*

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Central perforations of the tympanic membrane are a common condition. It has been variously estimated that from one to six million people in the United States suffer from this condition.

Closure of one of these perforations is very gratifying to a patient. He may gain as much as 25 db of hearing. His middle ear and mastoid are protected from infected material in the ear canal (during bathing, swimming, etc.). He has less chance of an otitis media during an upper respiratory infection, and in some cases an annoying tinnitus is relieved.

I wish to discuss in this paper the office technique for closing perforations and also to describe a skin grafting procedure for closure of tympanic perforations which has interesting possibilities.

ANATOMY.

The pars tensa of the tympanic membrane is approximately 9×10 mm. in diameter. It is attached peripherally to the annulus tympanicus and centrally envelops the manubrium. The blood supply comes from the anterior tympanic and deep auricular branches of the internal maxillary artery and from the stylomastoid artery. There is rich anastamosis of the blood supply as it courses from Shrapnell's membrane along the posterior border of the manubrium and from around the periphery of the drum.

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HISTOLOGY.

The drum consists of four layers:

1. Squamous epithelium (external surface).
2. Radially arranged connective tissue from the manubrium to the annulus.
3. Annular connective tissue.
4. Middle ear mucosa (inner surface) of cuboidal to pseudostratified columnar epithelium.

PATHOLOGY.

Perforations are caused by trauma (burns, blasts, punctures, etc.) or infections (otitis media).

Regeneration of an intact tympanic membrane during healing is quite common; in fact, the cause of a perforation's becoming permanent is poorly understood. Dunlap and Schuknecht[1] have noted that in permanent perforations the outer squamous epithelium grows around the edge of the perforation to contact the inner mucosal layer; thus, they feel, acting as a limiting membrane on the middle connective tissue layer.

THERAPY AND REVIEW OF LITERATURE.

Before any chronic perforation can close, the limiting epithelium must be destroyed. Early therapy of perforations overlooked this and consisted merely of applying prostheses of various types: Banzer[2] in 1840 used pig's bladder membrane stretched over an ivory tube. Yearsley[3] in 1842 used a cotton wool pledget; Toynbee[4] in 1853 used Indian rubber and gutta percha. In 1850 Clark[5] used rubber, and in 1876 Blake[6] used paper. In 1887 Guaranowski[7] employed egg membrane; Pollack[8] in 1882, and Isaacs[9] in 1925 used collodion. More recently, in 1951, Pohlman[10] developed Koragel inserts.

Some investigators have noted that patching perforations occasionally leads to an actual closure: Barclay[11] in 1890 used paper discs. Other reports of closure by mere patching have been made by: Nasiell[12] in 1934, and Steinmann[13] in 1945, using cellophane patches. In 1947 Unger[14] used gold foil and scarlet red ointment, and in 1951 Hall[15] reported closure of a fair percentage of perforations merely by applying a Yoshimo paper patch with 5 per cent celluloid in acetone as an adhesive.

The principle of cautery to destroy the limiting epithelium started with Roosa[16] in 1876, who used silver nitrate. Later Politzer[17] in 1895 also used silver nitrate, and Okuneff[18] in 1895 introduced trichloracetic acid. Joynt[19] in 1919 combined cautery with patching, and since then cautery with some form of patching has been used successfully by Stinson[20] (1941), Alexander[21] (1943), Fox[22] (1945), Ireland[23] (1946), Korkis[24] (1947), Folbre[25] (1947), Baron[26] (1948), Jordan[27] (1952), and Schrimpf[28] (1954).

The use of repeated cautery at roughly weekly intervals to prevent re-epithelization was used by Allport[29] (1916), Dunlap[30] (1917), Linn[31] (1944), Szpunar[32] (1947), Dunlap and Schuknecht[1] (1947), Adams[33] (1952), Derlacki[34] (1953), Peele[35] (1953) and Mullins[36] (1953). Derlacki[34] has noted that perforations can be closed by repeated cautery, which had failed to close by the cautery and patch method. This has also been our experience in our series of 65 perforations.

We feel that the cautery and patch method is successful where there is a fair blood supply, and destruction of the limiting epithelium is followed by rapid outgrowth of granulations, from the middle layer of the drum, which keep ahead of epithelization until opposing edges meet and seal with closure of the perforation.

Unfortunately, in many cases this exuberant granulation growth does not occur, and epithelization is able to circumvent and limit the healing edge of the perforation. This situation seems to arise when a drum is very scarred, and appears to have a poor blood supply.

METHOD.

Our technique is essentially the same as that of Linn[31] and Derlacki.[34] It consists of weekly cauterizations of the entire margin of the perforation with 50 per cent trichloracetic acid. This is applied with a thin wire applicator tipped by a tightly wound cotton ball about one mm. in diameter. The wire should be malleable, as it may have to be bent to maneuver in tortuous canals. The applicator is dipped in acid, blotted gently on a kleenex and applied to the edge of the perforation and peripherally for a distance of about one mm. A white eschar forms over this area. A thin cotton patch is then packed down against the drum and kept moist with Neomycin drops five mg. cc., two drops twice daily. Therapy is continued until Politzer inflation and pneumo-massage of the drum with the Seigle otoscope demonstrates that the drum is completely intact.

Before closure is started the perforation is measured with a fenestrometer and an audiogram is usually done, and is repeated after patching the perforation with cigarette paper. The resulting hearing gain has been found to be a reliable index of the final hearing level after the closure.

OBSERVATIONS DURING CLOSURE ROUTINE.

1. The first few treatments are painful. To obtain better cooperation, especially in children, we place a cotton patch moistened with Bonain's solution (*i.e.*, equal parts of cocaine, menthol and phenol) against the drum for 10 minutes before treating the ear. After the first few treatments this is usually not necessary.

2. Some perforations, even very large ones, respond promptly and close in a few weeks; others, equally large, quickly show an active border, but growth is sluggish and any missed treatments usually stop growth advance. An active border is thick and slightly hyperemic; it bleeds easily and turns white with the slightest touch of trichloracetic acid, even as weak as a 5 per cent solution.

In some markedly scarred drums, especially those caused by burns, trichloracetic acid will not form a white eschar. This indicates a long and halting course in the closure, probably because of a poor blood supply. In these we apply acid heavily, sometimes over the entire drum to destroy the scarred area and enlarge the perforation peripherally to a better blood supply. After months, an active border appears, and then narrowing of the perforation begins. It progresses haltingly, often with periods of temporary widening, and any missed treatments cause a loss of activity. Any attempt to change treatment, such as the use of a patch, or change to a weaker (5 to 10 per cent) trichloracetic acid has, in our hands, resulted in cessation of growth. Occasionally, after a very long time, these perforations have become very active and close rapidly. More frequently the final one-half to one-fourth mm. of the closure has been a prolonged process, with the perforation fluctuating in size and sometimes breaking open the week after it appeared closed. (All perforations should be followed closely for several weeks after they appear to be closed.)

3. Where therapy is discontinued before closure is achieved, scarring always gradually enlarged the perforation to almost its original size; however, where closure has occurred, this process merely thins the healed drum.

4. Besides Neomycin drops (5 mg. cc.) as a moistening agent for the cotton patch, we have used aqueous Furacin and 40 per cent alcohol. Others[1,31,34] have used Euthymol or 5 per cent urea in saline. Apparently no one drop is essential, but the cotton patch should not be allowed to dry. The functions of a moist cotton patch appear to be:

a. To prevent drying and crusting.

b. To provide a mildly stimulating irritation. Antisepsis is not too important; in fact the intervention of active infection has on several occasions activated sluggish or non-responding perforations to close rapidly. This has also been observed by Derlacki.[31]

c. If the ossicular chain is mobile, the moist cotton patch will cover the perforation and raise the hearing level. Our most faithful and appreciative patients have had bilateral perforations of this type. They will come back faithfully each week just for the hearing improvement which they receive by the cotton patch.

RESULTS.

From January, 1949, to May, 1954, we attempted to close 65 central perforations of the tympanic membrane. Total, marginal, and attic perforations, or the presence of cholesteatoma or squamous epithelium in the middle ear, attic, or mastoid were considered contra-indications to closure; however, no restrictions were made because of the size of the perforations, scarring, or adhesions to the promentory. All perforations had been present at least three months. Of the 65 perforations treated over this five-and--a-half-year period 57, or 88 per cent of them, were successfully closed. Four later reopened and of these, two quickly reclosed when treatment was resumed. A third case was closed by skin grafting, and the fourth case refused further treatment.

In a successful closure, how long a course of treatment may be expected? This table is a tabulation of all successful closures, showing the percentage closed at definite intervals:

	No Closed	Per Cent Closed	Total Per Cent
to 10 weeks	26	46%	
11 to 6 months	18	32%	78%
6 to 12 months	6	10%	88½
1 to 2 years	7	12%	100%

Thus, one-half of the closures occurred within 10 weeks and three-fourths of the successes occurred in the first six months, while 7 or 12 per cent took one to two years. We feel that several of our failures might have closed had they been as faithful as these seven cases.

ANALYSIS OF FAILURES.

An analysis of the eight failures shows that except for three (Nos. 6, 7 and 8), they were behaving in about the same way as did some of the similar perforations which later closed.

In our present state of experience we believe that with faithful return for treatment we would be perfectly justified in in-forming a patient that his chances of eventual closure by this method are very good (approximately 90 per cent).

These results in the office treatment of perforated eardrums are comparable with those of other investigators using this or similar methods. We have in general been quite satisfied; however, there are two rather major shortcomings of this method:

1. Very young children cannot be treated satisfactorily because some cooperation by the patient is necessary.

2. If treatment is prolonged, some patients tend to become discouraged or are unable to return at the regular weekly intervals which are so important to success.

Case	No. of Treatments	Orig. Size in Mm.	Size When Last Seen	Remarks
1	20	3x2	2½x2	Patient discontinued treatment
2	21	4x4	2 mm.	Patient discontinued treatment
3	50	6x5	½x½	Moved away
4	52	6x5	½x½	Discharged as hopeless after one year
5	56	6x5	2x2	At one time was ¼ mm. across, then patch applied
6	71	5x4	2x2	Always irregular treatments because of work
7	80	3x3	1x1	Has since closed with skin graft
8	80	6x5	2x2	Still under treatment; fluctuation

It was in seeking a solution to these problems that a trial at skin grafting perforated eardrums was made. We have found that the idea, as in most instances, is not new. Berthold[37] in 1878 and 1890 reported on successful skin grafting procedures to close perforated eardrums. He cut very thick grafts the same size as the perforation and of a thickness so that when laid on the promontory they would fit the perforation like a plug. Two of these cases were followed 14 and 15 months respectively. Eli[38] in 1880 reported on skin grafting the middle ear promontory in chronic otitis media, evidently for the purpose of reducing the secreting surface. His results were poor, and no closures were effected. Roosa[39] also apparently tried this procedure. Tangeman[40] in 1883 reported a partial closure from skin grafting a dry perforation after roughing the margins.

Since this study was started, House[41] in 1953 reported four

While the overall percentage of successful graft closures is less than closures by the office method, gradual improvement in our technique has improved our expectancy of successful graft closures considerably.

COMPARISON OF EARLIER AND RECENT RESULTS.

70 Graft Closure Attempts.	
1st 10 cases	1 success
2nd 10 cases	4 successes
Last 10 cases	10 successes*

* Four of these last ten tympanic perforations were very large—approximately 8x7 mm. in diameter.

EVOLUTION OF OUR PROCEDURE.

The actual technique of this procedure has gone through considerable evolution, as we sought to better our results; however, the basic principles of the operation are fairly simple. The outer layer of squamous epithelium on the drum is dissected off with a special chalazion-type curette. It is peeled off as a layer (see Fig. 1). Skin from one of several sources is then trimmed to the appropriate size and placed on this raw bed. This method, when successful, produces a nearly normal free-lying eardrum. It differs from other methods in that no attempt is made to provide a bed for the central portion of the graft, which overlies the perforation. Berthold,[37] and more recently House[41] and Zollner,[42] used the promontory as a blood supply for the central portion of their grafts. Their methods, however, would be inclined to produce adhesions to the promontory and to obliterate, more or less, the middle ear. The question arises as to why the central portion of our grafts can survive without being in contact with any blood supply.

Pagett,[43] Conway[44] and others have studied the manner in which a graft "takes." For the first 48 to 72 hours the graft is nourished, and the waste products are disposed of by a "plasmic circulation," which they feel is largely extra cellular by imbibition. From 16 hours on, there is proliferation of endothelial buds from the host's blood vessels. At 65 to 72 hours, continuity of host's and graft's blood vessels has been noted, and normal circulation in the graft is apparently largely complete and re-established by the eighth day. Preliminary fixation of the graft occurs within five hours by a fibrin clot. This clot gradually retracts and is invaded by leukocytes and phagocytes. On the fourth or fifth day fibroblasts begin to invade the clot, and organization is fairly complete by the tenth day.

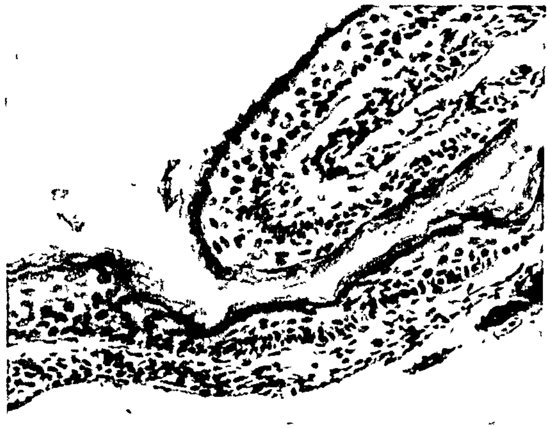

Fig. 1 Outer surface of tympanic membrane removed with a curette to make a bed for skin graft

The above studies would suggest that eardrum grafts survive the first 2 to 3 days on "plasmic circulation," supposedly extravascular. McLaughlin,[45] however, feels that much of a graft's circulation in the first few days is intravascular, because a few of the many small severed blood vessels of the graft come in contact with a few of the many severed blood vessels of the bed. We believe that this concept of intravascular circulation in the fresh graft gives a much more satisfactory explanation for the survival of the central portion of our eardrum grafts which overlap the perforation. The following study supports this contention:

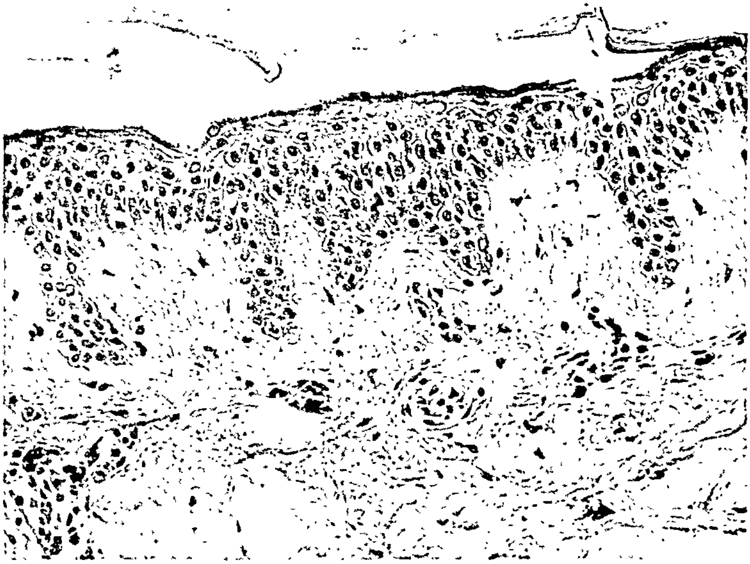

Fig. 2-A. Split thickness skin graft four hours after being placed on bed. Blood vessels are in spasm and contain no erythrocytes.

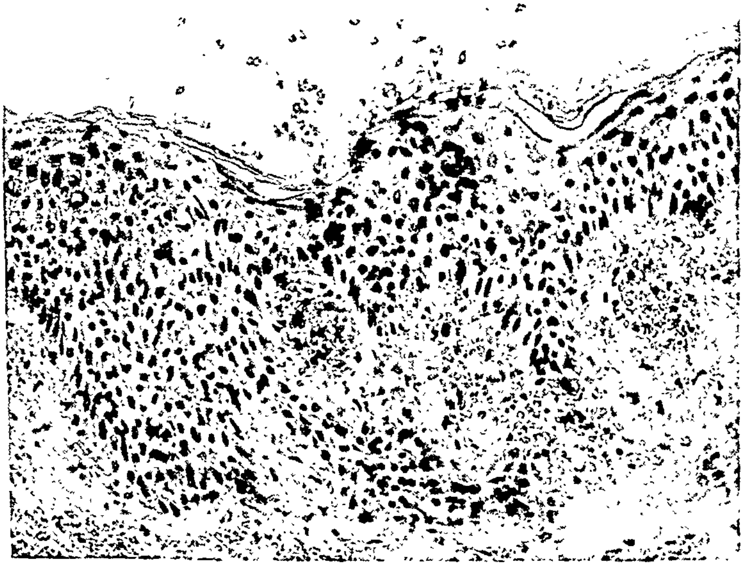

Fig. 2-B. Split thickness skin graft 24 hours after being placed on bed. Blood vessels are relaxed and dilated, and contain many erythrocytes.

Several split thickness skin grafts were cut and then laid back on their bed. After four hours microscopic sections showed an intense spasm of the graft's blood vessels, which squeezed out all erythrocytes (see Fig. 2). Microscopic sections of 24 hours' specimen showed, however, that the graft's blood vessels had relaxed, and all vascular spaces were filled with erythrocytes. This filling of the graft's blood vessels may have been from capillarity, or by continuity of graft and bed blood vessels. We believe it indicates some intravascular graft circulation.

In any case graft survival requires a good bed and a healthy graft. Atraumatic surgery is important, as is absence of infection. Good contact of graft and bed is also desirable, but we have found that too much pressure from packing may cause a slough of the central portion of the graft which has no underlying bed.

Many factors which affect results have been studied. It might be well to discuss them under separate headings.

1. *Infection*: We believe that several of our early failures were largely due to operating too soon (one or two weeks) after an otitis media. In spite of pre- and postoperative antibiotics, otitis media developed during the postoperative course of five cases. In each case, partial or total slough, or failure to seal the perforation resulted. At the present we feel that surgery should be delayed for at least one month after an otitis media. Sterile operating room technique is important. We also routinely use a sterile dressing technique for three weeks, and place the patients on antibiotics for one preoperative and six postoperative days.

Once a graft has healed, it may have surprising resistance to infection. One case has had at least four myringotomies done through the graft for acute suppurative otitis media. Each of these myringotomies healed after the infection had subsided.

2. *Anesthesia*: Local infiltration with 2 per cent Zylocaine, containing 1/50,000 Adrenalin. This may also be used topically on the middle ear mucosa. The addition of intravenous

sodium pentothal is usually necessary while the drum is being scraped.

3. *Exposure*: Exposure of the operative field is extremely important. The denuding of the drum, placing of the graft and packing must be done with exactness and can never be done blindly with success. In fact, adequate binocular visualization of all parts of the drum is essential, and magnification with the operating Loupe glasses extremely helpful. Occasionally, especially in children, the canal is both wide and straight, and the procedure can be done without an incision; however, we would attribute a fair number of our failures to working without enough exposure for binocular vision or magnification, because of a desire to avoid an incision in the ear canal.

Several methods of surgical exposure of the entire tympanic membrane are available:

a. A Shambaugh-type endaural incision and retractor can be used just as in an endaural mastoidectomy or fenestration. It is usually also necessary to cut a U-shaped flap from the posterior canal wall and to enlarge the bony canal posteriorly, superiorly and, occasionally, anteriorly. The flap has its interior incision about 3 mm. from the drum, and its pedicle on the floor of the canal. It is rolled down into the floor of the canal, where it is not in the way, and is replaced at the end of the operation.

b. In very tortuous canals where the anterior canal wall is the main visual obstruction, a U-shaped flap from the skin of the anterior wall can be made with the pedicle on the skin of the tragus. After the skin is out of the way the anterior wall bone is curetted or burred until the drum is visualized in its entirety.

c. A much simpler procedure, which we now use almost exclusively, is to curette the anterior canal wall with a very sharp curette, without elevating the skin. A sharp curette will cut through the skin and remove it without tearing it. The bony canal can then be enlarged and straightened by thinning the bone of the anterior wall. The new surface thus created can then be covered with a primary or, later, secondary skin graft. This method will often give good exposure of the entire drum without an endaural incision and never requires more than a small T-incision at the superior margin of the canal.

4. *Graft Bed*: This is prepared as atraumatically as possible with 2 and 3 mm. chalazion-type curettes. They are angled 15 degrees forward or backward so as to maneuver in the canal. The outer squamous epithelial surface of the drum is peeled off, beginning at the edge of the perforation. Microscopic sections of the sheet of tissue removed have consistently shown that all of the squamous epithelium is removed, along with equal thickness of connective tissue (see Fig. 1). There are no rete pegs, which probably accounts for the fact that the entire specimen comes off as one layer. The entire layer removed measures from 0.15 to 0.2 mm., and the flat epithelium measures 0.05 to 0.08. There are no glands present in any section.

In our early cases the drum was denuded only 2 mm. from the edge of the perforation, and the graft was cut to approximately the same size. Now the bed and graft are made at least as large as the entire drum, and in very large perforations the denuding process is continued up onto the canal wall. This increase in the size of the bed decreases the chance of a slough. The increase in the size of the graft relative to the perforation makes it less likely that the graft will slip far enough to expose part of the perforation, or will be pushed through the perforation.

5. *Cutting of Skin Graft*: Four sources of skin for grafting of perforations have been tried. It is very important to cut these grafts just before they are used, so that they will not dry out. We feel that placing them in normal saline is injurious.

a. *Split thickness skin graft from the inner surface of the upper arm* (25 "takes," 9 sloughs). Miscroscopic sections show that this graft has very little hair and sweat or sebaceous glands. The skin is thin, and only a medium thickness graft is available. Its tendency to curl is easily corrected by backing the graft with rayon or cigarette paper, using aureomycin ointment as an adhesive. The donor site is covered with surgical rayon, gauze and elastoplast. It heals consistently in ten days, when it is dressed for the first time. The danger of contamination and infection during bathing, etc., and the inconvenience of the dressing inherent in grafts from the thigh is not present here. The cosmetic effect is nicely hidden, because the donor site is on the inside of the upper arm.

b. Three-quarter thickness grafts from the mastoid area (22 "takes," 1 slough). These were all thick grafts. Microscopic sections show that this skin contains many sweat and sebaceous glands and quite a bit of hair. The graft should be taken from in front of the hairline. Several of our best results have long hairs growing from the drum. This skin has less tendency to curl and is easy to work with. The donor site is included in the ear dressing, and heals inconspicuously in ten days. We are now using this source almost exclusively.

c. Full thickness grafts from the post auricular fold (1 "take," 3 sloughs). House,[11] and Zollner,[12] used this source in their cases. The skin is very pliable and easy to work with. It has only a moderate tendency to curl and has softer hair and fewer sweat and sebaceous glands. We have not used this source extensively because Pagett[13] says, and we agree, that split thickness grafts "take" much more readily than full thickness grafts. There is certainly a good deal of additional trauma in trimming the fat from the under surface of full thickness grafts.

d. Pedicle flaps from the ear canal, both delayed and immediate (no "takes," 4 sloughs). There is considerable technical difficulty in swinging such a flap; also, a large flap necessarily has a small pedicle and any packing would, of course, crease the pedicle and cut off circulation. The skin, however, has a very fine texture and does not tend to curl. These poor results may be partially due to the extremely large perforations which were attempted by this method; also, sea sponge packing was used in all four flap cases. In six of the seven cases where sea sponge packing was used, slough occurred.

6. Graft Placement: If there is any tendency for the graft edges to curl, it is backed with surgical rayon, using aureomycin ointment as an adhesive. The graft is then cut to the appropriate measured size with a sharp knife. If it is to extend very far up onto the canal, the peripheral edges must be serrated with a sharp knife to allow for folding of the edges of the graft. The graft is then gently picked up with fenestration forceps and placed near the drum. It is then teased into position with forceps, fenestration picks, ring curettes, or mastoid searchers. After it is apparently in place the edges are momentarily rolled back with a fenestration pick to assure eversion and to see that all the perforation is well covered. The graft is then covered with cigarette paper and a cotton pack.

7. Packing: It is important to get good contact between graft and bed. With this in mind, we first tried tight vaseline gauze packs, or sea sponges. These tight packs had a tendency to push the graft through the perforation into the middle ear. We also developed a feeling that the part of the graft overlying the perforation was more likely to slough when a tight pack was applied (as mentioned before, a tight pack might obliterate the "plasmic circulation" to this portion of the graft).

Later we dispensed entirely with packing and depended upon the contracting of the underlying elastic clot to bring the graft into contact with the bed. On a few of these cases the graft slipped so as to expose part of the perforation, or failed to seal, and we have now settled on the method of packing used by Rosen[16] in the stapedial mobilization operation. After the graft is in place, it is covered with sterile cigarette paper strips, which extend up onto the canal, and very thin cotton balls are used to hold the graft gently in contact with the bed. After the first layer, aureomycin ointment is mixed with the cotton to protect from infection and to make the pack easier to remove.

8. Postoperative Routine: The patient is kept on antibiotics for six days. The packs are removed entirely on the seventh day. The cigarette paper and debris are wiped out, under sterile precautions, on the fourteenth day. If raw areas are present on the canal, they are at this time covered with a secondary split thickness skin graft from the inner surface of the upper arm.[47]

The patient is Politzerized at one month.

We think it is a mistake to delay the complete cleansing of the canal and the Politzerization beyond the time specified. No harm can be done to a healthy pink graft and, if there is weakness, partial slough or failure to seal, it should be discovered as early as possible, as a regrafting procedure is best done as early as possible while the blood supply of the bed is still stimulated.

REGRAFTING PROCEDURES.

Fourteen of the failures were regrafted, with nine of these attempts leading to successful closure.

1. Four of the regrafting attempts were made in 1952. Two were done by split-thickness grafts from the arm, and two by rotation flaps from the skin of the ear canal. All failed.

2. The other ten regrafts were all done during 1955. Eight of these were successful, and the one failure had a third attempt at grafting, which was finally successful.

We have a general belief that regrafting of failures is perhaps more likely to succeed than the first graft, and think that regrafts should be done as soon as it is obvious that the first graft has failed. There is a great stimulation to the blood supply of the bed for a few weeks. It is probably best to remove the old graft entirely, freshen the bed, and apply an entirely new graft; however, three of our successful cases were done by a different method. The old graft was partially rolled back and the exposed bed freshened. A new graft was applied to this bed and laid against the rolled-back surface of the old graft. The edges of the two grafts were carefully everted and held in place with a soft cotton ball pack on cigarette paper.

ANALYSIS OF POSSIBLE CAUSES OF FAILURE.

In most of the 30 grafts which failed, it was believed that the factor causing the failure could be identified. Surprisingly enough, there did not seem to be any apparent correlation between the size of the perforation and either success or failure. The 56 perforations grafted varied from 3 × 3 mm. to 8 × 6 mm. The average size was approximately 4 × 5 mm. We consider that over a larger series of cases the smaller perforations should show a better chance of successful closure by skin grafting.

1. Four grafts "took" but did not seal in one quadrant:

a. Two of these were obviously from infection in the postoperative period.

b. The other two were in the same patient, and were ostensibly from blowing off the graft by Politzerization two weeks after surgery. We now wait one month before Politzerizing.

2. Five grafts "took" but slipped enough so that the perforation was not entirely covered.

a. Three of these were partly pushed into the middle ear, probably by a combination of too tight a pack and too small a graft.

b. In the other two cases the graft slipped enough to expose part of the perforation. No pack was used in these cases, which may have been responsible for the slipping.

3. Seventeen grafts partially or totally sloughed.

a. Five of these were obviously from infection.

b. In six, sea sponge packing was possibly responsible (as mentioned previously, six of the seven cases where sea sponge packing was used resulted in a slough).

c. In six cases there was no apparent cause for the slough.

4. Four grafts were late failures (the graft "took" but several months later, re-perforated). Two of these were caused by trauma, one by an otitis media, and one was apparently spontaneous.

LATE APPEARANCE OF GRAFTS.

The successful graft at first appears thick and hyperemic. It is sealed quite tightly at three weeks, and at six months is hard to remove. After the first year it gradually thins and becomes almost indistinguishable from a normal drum.

In one of our cases where no packing was used the graft "took," but slipped so as to leave a small slit of perforation still uncovered. Six months later this graft was removed and replaced with a fresh one, which was completely successful.

The old removed graft was a three-quarter thickness graft from the skin over the mastoid process. When it was removed with a curette the bed bled slightly. Microscopic sections through this graft showed that it was approximately the same (1.3 mm.) thickness as when it was cut six months earlier. There were still some sweat glands and a few hair follicles remaining. It was well vascularized, and there was almost no inflammatory reaction. The squamous epithelium was slightly flatter, but there were still rete pegs. At the periphery of the graft the squamous epithelium had grown around the edge and slightly underneath it, until it contacted and fused with the squamous epithelium of the surface of the drum. The area of attachment of the graft to the drum was quite vascular. There was mucosal lining which had grown across the under surface of the graft, where it overlay the perforation.

HEARING IN SUCCESSFUL GRAFT CLOSURES.

Twenty-five patients had hearing improvements of 10 to 30 db. Five failed to gain. In none was the hearing made worse. Ten patients were not tested. It was suspected that thick grafts would not give as good hearing as thinner ones. In two such cases the patient failed to make as much gain from grafting as from preoperative patching with cigarette paper; however, later as the grafts thinned, hearing came up to the expected level.

REPORT OF TYPICAL CASES.

1. Miss L. S., age 26, was seen in March, 1949, because of hearing difficulty all her life. Examination showed bilateral dry perforations; right 5x4 mm., left 4x3 mm. Closure routine was started. The left perforation closed in eight treatments, the right in 11 treatments Hearing gain *right, 28 to 12 = 16 db, left 28 to 8 = 20 db*

Remark This case illustrates how easily perforations sometimes close

2 Mrs M J G, age 27, was seen in February, 1954, complaining of an intermittent right otorrhea since childhood Seven years previously she was advised to have a radical mastoidectomy Examination showed a dry central perforation in the right tympanic membrane 5x4 mm Mastoid X rays showed no bone destruction A patch increased the hearing for the speech frequencies in the right ear from a level of 23 to 3 db = 20 db gain *Closure treatment was started, using neomycin drops as the moistening* agent After the seventh treatment an active margin was noted except along the handle of the malleus The entire margin was active after the eleventh treatment, at which time the perforation began to narrow slowly Just after the thirteenth treatment the patient developed a severe painful external otitis and otitis media, with severe swelling A pseudomonas aeruginosa was cultured from the drainage Closure routine was stopped, and the patient put on aerosporin eardrops and codeine for pain Ten days later when the infection subsided, no perforation could be demonstrated *Hearing gain, 23 to 3 db, exactly as predicted by patching* The drum now appears normal

Remark This case illustrates the beneficial effect of intercurrent infection on closures

3 Mrs W A C, age 31, was seen in March, 1952, complaining of bilateral otorrhea intermittently with colds since childhood Examination showed *bilateral dry perforations in both drums right, 5x4 mm, left,* 6x5 mm The left drum was very scarred and the malleus handle was protruding into the perforation Bone conduction was normal Patching caused a hearing gain of 25 to 5 = 20 db, right ear, and 32 to 8 = 24 db, left ear Closure routine was started using neomycin drops Over the next two years the patient received 90 closure treatments in the left ear and 72 in the right ear During the first year these treatments were broken up *by an attempt at patching, a trial of weaker (10 per cent)* trichloracetic acid, and finally an attempt at skin grafting the perforations All failed It was noted that missed treatments caused the perforation edges to become inactive Eleven months after treatment was started, the perforations measured right, 5x4 mm, left 4x3 mm After this time, there was no variation from the usual weekly painting with 50 per cent trichloracetic acid, cotton patch and neomycin drops The *right perforation was closed after 20 months and 72 treatments, the left* closed after 24 months and 90 treatments Hearing gain right, 25 to 2=23 db, left, 32 to 17—15 db

Remark This case shows how prolonged a successful closure can be After one year of treatment these perforations were almost as large as they were before treatment was started, yet both were successfully closed Few patients would return this faithfully

4 Miss W K age 12, was seen August, 1951, complaining of intermittent otorrhea all her life Examination showed a draining right ear with a 3x3 mm central perforation of the right tympanic membrane Culture revealed a pseudomonas aeruginosa infection, which cleared on neomycin drops *The patient then had 80 weekly treatments for closure,* during which time the perforation fluctuated in size, on one occasion being only one half mm in diameter She then became discouraged and discontinued treatment in July, 1953 When next seen in June, 1955 the perforation was as large as before treatment was started On June 21, 1955, a split thickness graft from her arm was used to cover the perforation It took"

well and an audiogram three months later showed a hearing gain of 25 to 15=10 db. It is hoped that this will continue to improve as the graft thins.

5. Mrs. W. R. W., age 19, was seen June 11, 1954. Examination showed a dry 6x6 mm. central perforation of the right tympanic membrane. On June 24, 1954, via an endaural incision, the perforation was grafted with a split thickness graft from the upper arm. The graft "took" well and is intact 15 months later. It has thinned to approximately the same thickness as the rest of the drum. Hearing gain, 17 to 7=10 db.

CONCLUSION.

While one-half of these successful skin graft closures have been followed less than nine months, successful graft closures have been followed two and three years. So far, only a very few of the procedures have become a failure later than six weeks postoperatively. None, so far, have developed as failures later than four months postoperatively. It is hoped, therefore, that the long term results reported for these skin graft closures will continue to hold up. We realize that the office method of closing tympanic perforations is a well-proven procedure, while much has still to be learned about skin graft closures; however, our results with skin grafting are encouraging, and the advantages of grafting are considerable in dealing with children and where a prolonged course of office treatments is anticipated. We feel justified in recommending this procedure to any patient when weekly office cautery has not made progress toward closure within ten weeks. We also feel that in the event a graft is unsuccessful, a second, and if necessary a third graft can be advised.

SUMMARY.

Closure of 57 of 65 central eardrum perforations by weekly cautery with trichloracetic acid is reported.

Closure of 40 of 56 perforations by skin grafting is reported.

The literature on this subject, and factors affecting results are discussed.

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SEVENTH INTERNATIONAL CANCER CONGRESS—LONDON, ENGLAND.

The Seventh International Cancer Congress will be held in London, England, July 6-12, 1958, under the Presidency of Sir Stanford Cade. Congress headquarters will be The Royal Festival Hall. This Congress is sponsored by the *International Union Against Cancer.*

There will be two main sessions of the Congress: A. Experimental; B. Clinical and Cancer Control. Special emphasis ill be placed on hormones and cancer, chemotherapy, carcinogenesis and cancer of the lung.

Papers will be considered only if submitted with an accompanying abstract (not over 200 words) before October, 1957, and if dealing with new and unpublished work.

The registration fee for the Congress will be £10 (ten pounds) or $30, and the latest date for registration without late fee will be January 1, 1958.

Registration forms and a preliminary program will be available early in 1957 on application to The Secretary General, Seventh International Cancer Congress, 45 Lincoln's Inn Fields, London, W. C. 2, England.

EDUCATIONAL GUIDANCE FOR PARENTS OF YOUNG DEAF CHILDREN.

EDGAR L. LOWELL, Ph.D.,
and
GEORGINA RUSHFORD, M.A.,
Los Angeles, Calif.

Ten years ago THE LARYNGOSCOPE published an article from the John Tracy Clinic entitled, "What the Otologist Should Know About the Educational Problems of Deafness." To quote from the article:

> "To sum it all up the doctor should be able to offer constructive advice to the patient he cannot help medically. He should have at hand concise, accurate and definite information as to what may be done for such a patient—child or adult. He should know where the patient may go for help. He should know what local and national organizations offer facilities for the hard of hearing and the deaf.*"

A decade later we are able to report how well this need is being filled. As part of a larger study carried on to evaluate the success of the Correspondence Course distributed by the John Tracy Clinic, we have collected factual information on the kind and amount of educational advice given to parents of young deaf children.

At the John Tracy Clinic, where we have been primarily concerned with educational guidance, we are constantly impressed with the importance of the educational information given by the doctor. At the crucial time of interpreting the diagnosis of hearing loss to parents, prompt and positive suggestions do much for the future adjustment of the family and child.

The main intent of the larger study was to evaluate the Cor-

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* Harriet Montague: What the Otologist Should Know About the Educational Problems of Deafness. The Laryngoscope, Vol. 56, No. 6, June, 1946.

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respondence Course which the Clinic offers to parents of young deaf children all over the world. The course was first sent out in 1943, and during the 13 years since then more than 7,000 families have been enrolled for these lessons, from approximately 52 different countries. The course covers one year of work that any untrained parent can do at home without supervision except that supplied by mail from the Clinic. The program is explained in simple language, and it is necessarily flexible since the children range in age from 22 months to 5 years. The idea is to provide suggestions easily understood and easily followed, to give the parents something constructive to do to help their deaf children, and to give the children themselves something constructive to do to utilize their energies and natural abilities, thus putting them into communication with the world around them.

The material on which these figures are based comes from the first 5,000 families enrolled in the course. The course has been sent to all parts of the United States and has had a geographical distribution approximately the same as the total population of the United States.

At the time of parent application 854 of the children had [illegible] had an accurate evaluation of the extent of their hearing loss; 1504 had been classified as hard of hearing, [illegible]age losses up to 75 decibels through the speech range, and 2645 had been classified as severely or profoundly deaf with losses greater than 70 decibels. The majority of these parents became aware of their children's deafness through the child's failure to respond to sound. Specifically, in 40 per cent of the cases it was failure to respond to the parent's voice, and in 23 per cent of the cases failure to hear loud environmental sounds. Failure to develop speech at the normal time accounted for 26 per cent, and the remaining 6 per cent became aware of the loss only at the time of the doctor's diagnosis.

The importance of the doctor's role in providing such information is emphasized by the fact that 92 per cent of the parents in this study had no previous knowledge about deafness and the education of the deaf. Seventy-one per cent of this parent-group indicated they had received advice from the physician concerning their deaf child's education. Many years ago the importance of beginning the specialized education required by a deaf child at the earliest possible moment, was not recognized. The fact that the average age of the children in this sample was 3½ years indicates the medical profession is now cognizant of this trend and is working to support it.

Seventy-seven per cent of the doctors discussed the teaching of speech with the family. Some few parents still erroneously believe that the manual method, that is, finger spelling and signs, is the only avenue of communication possible for the deaf person. To know that a child can acquire language and speech is important in helping a family adjust to the situation.

The questionnaire on which most of this information was based did not specify whether the doctor who provided the educational information was an ear specialist, a general practitioner, or a pediatrician; in fact, only 715 of the families reported they had received specialist treatment at the time of application for the course. This evaluation of the medical recognition of educational problems of deafness reflects knowledge on the part of the entire profession and not specifically on the part of otologists. We feel this is a great compliment to the entire profession.

It is unreasonable to expect the doctors to provide prolonged guidance; however, they are making this possible by putting parents in touch with organizations specifically designed to provide educational follow-up. Seventy-one per cent of the doctors knew the names of the schools for the deaf nearest where the family lived and referred them to other organizations offering help. Sixty-two per cent gave the families specific help with additional sources of information. This examination of the Clinic's records indicates that 40 per cent of all applicants for the Correspondence Course were referred by the medical profession.

Further evidence of the close cooperation between the medical and educational fields comes from the fact that only 4 per cent of the doctors were reported as still using the old-fashioned and incorrect term, "deaf and dumb" in discussing children who are only auditorily handicapped. This antiquated term has unpleasant and misleading connotations for the parents and the general public.

Totally, these figures indicate that doctors have been doing an excellent job with a difficult but essential educational problem.

A SPECIAL POSTGRADUATE COURSE.

An introduction to fundamentals of reconstructive surgery of the nasal septum and external pyramid will be presented by Yale University School of Medicine Department of Surgery, Section of Otolaryngology, in cooperation with The American Rhinologic Society and Grace-New Haven Community Hospital, May 11 - 18, 1957.

The course will be given under the guest direction of Dr. Maurice H. Cottle. It will consist of didactic lectures, case presentations, cadaver dissections, and observation of operations. All sessions will be held at the Yale-New Haven Medical Center, New Haven, Connecticut, U. S. A.

Enrollment will be limited. Applicants must be diplomates of the American Board of Otolaryngology, or its equivalent for doctors from other countries. Scholarship assistance is available to a limited number of foreign applicants.

For further information write to Assistant Dean of Postgraduate Medical Education, Yale University School of Medicine, 333 Cedar Street, New Haven 11, Connecticut, U.S.A.

HEARING LOSSES IN CLEFT PALATE CASES.†

The Incidence, Type and Significance.

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New York, N. Y.

PART I. NATURE OF THE STUDY.

Each patient with a cleft palate referred to the Speech and Hearing Clinic at Lenox Hill Hospital is given a complete pure tone audiometric test as part of the preliminary workup before speech therapy is begun. The susceptibility of individuals with this organic involvement to various types of upper respiratory conditions with its frequent effect upon the hearing mechanism has convinced us of the need for a careful investigation of the hearing of all cleft palate cases.

The audiometer used in conducting these tests was a Sonotone AE21 model. The testing was done in a sound treated room where quiet conditions prevailed throughout the course of the study. Threshold was established at the following frequencies: 125 c.p.s., 250 c.p.s., 500 c.p.s., 1000 c.p.s., 2000 c.p.s., 3000 c.p.s., 4000 c.p.s., 8000 c.p.s., 10,000 c.p.s., and 12,000 c.p.s. Whenever a loss of 25 db or greater was found by our air conduction tests, a bone conduction test on the affected ear or ears was performed. The definition of a hearing loss used in this study is a loss of 30 db or greater at one or more of the frequencies tested. We have chosen this intensity level because in many cases young children do not respond to pure tones the instant they hear them but wait until the sounds are clearly audible before giving evidence of having heard them. Thirty-five cleft palate cases were used in this study, ranging in ages from three to 23 years of age. As to the validity of audiometric tests on children three to five years of age in this study, we are convinced that after elicit-

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† Research conducted in conjunction with clinical work at Lenox Hill Hospital and submitted as part of the requirements for the Seminar in Speech Pathology at Brooklyn College under the supervision of Dr. Robert W. West and Dr. John K. Duffy.

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ing the cooperation of the child it is possible to perform an accurate pure tone audiometric test on these children as shown by the consistency of results found in our retests of children in this age group.

PART II. FINDINGS.

The type of hearing loss found in the cleft palate cases tested is predominantly conductive; that is, a loss caused by some impairment of the sound conducting mechanism responsible for the transmission of sound waves to the inner ear. Although none of the cases tested had pure preceptive losses, there were five mixed losses found among the group. One was a child with congenital atresia of the right ear, and our hearing test showed a severe conduction loss (60-65 db) throughout the range tested with slight nerve involvement. Three cases of mixed loss showed a history of chronic suppurative otitis media in which the infection may have spread to the inner ear causing some nerve involvement. Another case having a mixed loss showed nerve involvement principally at 4000 c.p.c. with the remainder of the loss conductive in nature. This patient sustained possible traumatic injury to the head.

Of the 19 cases of conduction hearing losses found, there were 12 instances of conduction losses affecting the high frequencies only, and probably due to the presence of lymphoid tissue in the naso-pharynx. There were seven cases in which there was a conduction hearing loss throughout the range tested. The results of the study are summarized in the Table on p. 1494.

It will be noted that in Part III of the table, which gives a breakdown of the range of frequencies involved in the 19 cases of conduction hearing losses found, no percentages are listed, and the total number of the cases is 21, rather than 19. This is because some of the cases of bilateral hearing losses showed a different type of conduction loss in each ear and were, therefore, mentioned in two of the categories. For example, in the case of a patient having a high frequency conduction loss in one ear and a loss throughout the range in the other, the first type of conduction loss was listed in Part IIIa, the second in Part IIId.

Classification	Number of Cases	Per Cent of Total Cases
Number of cleft palate cases tested	35	100%
I. Of the total number of cases tested, those having:		
a. no hearing loss	16	45.71%
b. some hearing loss	19	54.29%
		Per Cent of 19 Cases Having Some Loss
II. Of those cases having some hearing loss, those with:		
a. pure conductive loss	14	73.68%
b. pure preceptive loss	0	0%
c. mixed loss	5	26.32%
III. Of those cases having pure conduction losses, those with:		
a. high frequency conduction loss in one or more ears	12	
b. low frequency conduction loss in one or both ears	1	
c. conduction loss in one or both ears at very low and very high frequencies only (128 c.p.s., 256 c.p.s., 10,000 c.p.s., 12,000 c.p.s.)	1	
d. conduction loss throughout range tested	7	

PART III. SIGNIFICANCE.

The high incidence of conduction losses of hearing in cleft palate cases may be due to the presence of the cleft in the hard or soft palate which permits the entrance of organisms producing upper respiratory infections, chronic colds and influenza, otitis media, etc. In addition to this factor, the incomplete closure of the naso-pharyngeal port caused by an unusually short velum which does not make contact with the superior pharyngeal constrictor is another means by which the organisms producing upper respiratory conditions can gain access to the body.

In a large number of cleft palate cases, the hearing loss appears to be caused by the presence of prolific lymphoid tissue in the naso-pharynx which occludes the mouth of the Eustachian tube and interferes with the proper ventilation of the middle ear. The presence of this condition has been determined by direct and indirect naso-pharyngoscopy on some of our cleft palate cases having conductive losses of hearing. The removal of this lymphoid tissue is frequently contra-indicated in cleft palate cases (as is the removal of the tonsils) because any tissue in the area of the oro- or naso-pharynx seems to help the child to achieve the closure of the naso-pharyngeal port so necessary for the development of intelligible speech.

Dr. Fred S. Dunn, Chief of the Oral Surgery Division at Lenox Hill Hospital, suggests as an explanation for the high incidence of hearing losses among cleft palate children, the practices of some cleft palate surgeons who, during an operation to close the soft palate, frequently produce scarring or distortion of the tissues at the naso-pharyngeal orifice of the Eustachian tube interfering with its normal function.

The type of conductive loss found most frequently in cleft palate cases is one which affects the very high frequencies first (8000 c.p.s. to 12,000 c.p.s.) and then, if the cause of the hearing loss is not discovered and proper medical care administered, the entire range, resulting in the reduced acuity for all speech sounds characteristic of advanced conductive losses. The frequency of high tone losses with no nerve involvement found in these cases tends to support the findings of Crowe[1,3] who stated that high frequency losses can be purely obstructive in nature.

This type of conduction loss, in its initial stages, does not affect the ability of the patient to comprehend speech because the frequencies affected are above the range important for the understanding of speech. Its significance lies in the fact that it is frequently the precursor of a more serious conductive loss and indicates the necessity of an immediate ear, nose and throat examination to determine the cause of the condition.

(We have found that conductive losses in children who do not have a cleft palate also tend to affect the high frequencies first, and then, if proper medical care is not administered the entire range of hearing.)

Because this type of conduction loss affects frequencies above the range important for understanding speech, speech reception tests such as the fading numbers test currently used in many public school systems and group pure tone audiometric tests and screening tests using frequencies only in the speech range will not be successful in discovering this incipient type of conductive loss at the time when a proper otological check-ups might arrest the condition. Only a complete, individually administered, pure tone audiometric test which tests frequencies above and below the speech range (125 c.p.s., 250 c.p.s., 10,000 c.p.s., and 12,000 c.p.s.) will detect this type of conductive loss. The public school systems, the hospital and university speech and hearing clinic, and all agencies working with large numbers of children should recognize the diagnostic significance of this prodromal conductive loss in cleft palate children and provide for adequate medical treatment through the services of a competent otologist. Proper medical treatment will frequently be successful in stabilizing and even eliminating the hearing loss and certainly in preventing later deafness in adulthood.

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In Memoriam

ISAAC HAMPSHUR JONES, M.A., M.D.,

1881-1956

Isaac H. Jones was born in Philadelphia in 1881. He attended Amherst College, from which he graduated Phi Beta Kappa. In 1906 he received his M.D. degree at the University of Pennsylvania School of Medicine, with election to Alpha Omega Alpha. In his early years of otologic practice he came under the spell of Robert Barany, whose "epochmaking" work on vestibular nystagmus, a short time before, had focussed the attention of otologists the world over. A new field was opened up, and Isaac Jones entered it with enthusiasm. In Philadelphia he had as guide and counsellor, Dr. B. Alexander Randall in otology, Drs. Charles K. Mills and T. H. Weisenburg as neurologists, and Drs. Lewis Fisher and Seth A. Brumm as fellow-workers.

By 1916 he had become a recognized leader in the otologic quest, and was invited to address the Academy of Medicine in Washington, D. C., on the ear and aviation. In his audience was Col. Theodore Charles Lyster. Later, after this country entered World War I, Dr. Lyster brought Dr. Jones into Army service and introduced him to his uncle, Major-General William C. Gorgas. In May, 1917, Lyster, Jones and Eugene R. Lewis formulated the "Physical Examination for Flying," which was made official for the Army as A.G.O. 609.

Under Col. Lyster, now Chief Surgeon of Aviation Section, Signal Corps, United States Army, Drs. Jones and Lewis were appointed to organize boards of medical exam-

iners of candidates for commissions in this new service. The two men travelled from one recruiting center to another, from the Atlantic seaboard to the Pacific coast, setting into activity 35 medical aviation boards.

Overseas in 1917 and 1918, he had intimate contacts with British, French and American flyers, as medical counsellor, observer and—pupil. Again in France, in 1919, at the Versailles Peace Treaty, Col. Jones was appointed medical advisor for aviation in the American delegation, and was one of the two American members of the committee which drafted the International Requirements for Commercial Flying. In recognition of his services the Government of France created him *Chevalier de la Légion d'Honneur.*

In the interval between his periods of overseas service he edited the *Air Service Medical,* with the collaboration of Frank L. Dennis, William F. Patten and Eugene R. Lewis; he also served for a time as editor of *Aviation Medicine.*

His *magnum opus,* "Equilibrium and Vertigo", the fruit of his clinical work prior to the war, and dedicated to his three mentors, Drs. Mills, Randall and Weisenburg, was published in 1918. In the preface he gives abundant credit to his collaborators, Lewis Fisher, Eugene R. Lewis, George E. Shambaugh and others, as well as a gracious tribute to his wife's assistance. (Dr. Fisher, named as co-author, wrote the section "An Analysis of Pathologic Cases", an account of clinical findings and their interpretation, in cases under observation from 1914 till 1917.)

By a happy circumstance (may we not call it a "fortune of war?") he was able to make an eleventh hour addition to the book's already rich contents, namely: Chapter III—"The Ear and Aviation". This brought formal acceptance by the Army, as shown on the title-page: "Adopted as Standard for Medical Division, Signal Corps, Aviation Section, by Surgeon General, and Chief Signal Officer, U. S. Army." This book may, in future years, prove to be a treasury of material for students of the history of medicine. (The same, in lesser degree, is to be said of his "Flying Vistas", published 19 years later.)

Leaving Philadelphia in 1920, he began a new practice in Los Angeles in association with Drs. T. C. Lyster, Samuel D. Ingham and Eugene R. Lewis and soon gained prominence in his field.

A common interest in acoustics brought together Prof. Vern O. Knudsen of the Department of Physics in the University of California at Los Angeles and Isaac Jones, clinician-otologist. Their joint labors resulted in a series of articles on hearing problems, the creation of an early audiometer and of an experimental hearing-aid. He was appointed Research Fellow in Zoology at the University; Dr. Walter P. Covell and he conducted animal experimentation to determine the effects of vitamin deficiencies in rats—for example, changes in general nutrition and the degeneration of spiral ganglion cells resulting from withholding of yeast from the diets.

In another "Venture to the Interior" (to borrow a title from Laurens Van der Post) Drs. Ingham, I. Leon Myers and Jones studied in slow-motion pictures, the actions of cats dropped, with feet up, from a height of five feet to a mattress—normal cats; others with surgical lesions of the labyrinth and the cerebellum. Normal animals turned in the air in the first two feet of the fall and landed on their feet. Those with vestibular lesions fell without showing any tendency to right themselves. Dr. Ingham writes that "the results clearly demonstrate the major importance of vestibular function in the control of the cats' marvelously coordinated movements."

The significance of this study is shown in a section of Dr. Jones' last book, "Flying Vistas, the Human Being as Seen Through the Eyes of the Flight Surgeon," published in 1937. The volume tells the story of Aviation in World War I, and is dedicated to Gen. Theodore Charles Lyster. It was the duty of Drs. Lyster and Jones to organize the new Aviation Section, as already told. In the book we follow the course of its growth and the experiences of the two men in this country and in France. In its earlier chapters "it attempts to explain some of the mysteries of the human body, including the difficult things about the special senses . . . in plain English." In the chapter on Blind Flying, Dr. Jones sets forth the lessons of the "cat" experiments and their application "when we consider the illusions that come from the ears . . . when we are flying blind." The author's style is light-hearted, revealing and sensitive. The history-making achievements of the Aviation Section are told, both gaily and with pathos. Any present-day flyer will do well to read this book, which won high praise from officials of the Aviation Corps. Of the Flight Surgeons of 1917-18 Dr. Jones was the first to receive a commission. In the words of a fellow-officer, "no one is better qualified to tell the tale of those thrilling days."

Dr. Jones was a prolific writer. Of his articles on acoustics many were the fruit of his association with Prof. Knudsen. His work on vestibular problems resulted in many contributions of weight, of which one in particular is chosen for special mention here—"Vestibular Experiences". It contains a detailed report of the case of a boy of 15 with "complete vestibular block . . . perfect auditory function and no vestibular function;" he and Dr. William G. Spiller had presented their findings in this case before the American Neurological Society* and he later reported it to the American Laryngological, Rhinological and Otological Society**: "Dr. Spiller . . . for the first time revealed the Central Vestibular pathways," a demonstration of the vestibular tract in the brain-stem which "was made possible by a sequence of events that may never happen again." This article includes also an account of vestibular damage from streptomycin; the discussion which followed its reading is unique in that one of the participants, Dr. Samuel D. Ingham, Dr. Jones' close friend of many years, gave a neurologist's review of his own off-balance experience, being himself a victim.

Nutrition and Related Problems was the subject of many articles, including a long series of editorials published annually in THE LARYNGOSCOPE. In most of these essays he had as co-workers Eugene R. Lewis and others, including Leland G. Hunnicutt, Jerome A. Hilger and the writer of this obituary, who had the privilege of taking part in the work through a period of 15 years. It was his delight to inject here and there, when an opportunity came, a witticism or a timely quotation from an old-time author. Always, simple English was his goal; "let us make it shorter and so plain that A or B will get the meaning." His appraisal of material was sound. Those who worked with him were the richer for so doing.

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*Isaac H. Jones, M. D., and Wm. G. Spiller, M. D.: "The Central Tracts of the Nervous Vestibularis." Brain, Sept., 1925.

**Isaac H. Jones, M. D.: "Vestibular Experiences." Trans. of Amer. Laryngol., Rhinol. and Otol. Soc., Inc., 1949.

In 1922 Dr. Jones enlisted the interest of other Los Angeles otologists and brought Prof. Robert Barany to this city for a course of lectures. (He often said whimsically: "Eleven were invited from other States, and of these 13 came.") Similar courses followed in the ensuing years. In 1928 the group took a long step forward in the formation of the Research Study Club of Los Angeles. An early major activity was a series of lectures, given by local otologists, neurologists and neuro-surgeons, and later combined in one volume, "Studies of the VIII Nerve, by the Research Study Club". In 1931 began the Midwinter Clinical Course, the first gathering being for otologists. Its success was so inspiring that for the following Convention in 1932 the ophthalmologists, led by Dr. A. Ray Irvine, joined forces with their colleagues. Thus came into being the annual Midwinter Clinical Course for the combined specialties. The Club was now incorporated and Dr. Jones continued to act as chairman of its Committee, an office he had occupied from the beginning and was destined to hold until 1955. Guest lecturers have been brought from Great Britain, Austria, Switzerland and other Europen countries; from India, from many medical centers in this country, and from Canada.

Dr. Jones enjoyed his membership in the following socities: Diplomat certified by the American Board of Otolaryngology, Los Angeles County Medical Association—President of the Eye and Ear Section in 1937; American Medical Association; American Laryngological, Rhinological and

Otological Society, Inc.—Vice-President (Chairman of Western Section), 1923; American Otological Society; Association of Military Surgeons of the United States; Pacific Coast Oto-Ophthalmological Society; American College of Surgeons; American Academy of Nutrition—member of the Editorial Board of its Journal of Applied Nutrition; Editorial Board of THE LARYNGOSCOPE; Research Study Club of Los Angeles—chief organizer and chairman of its Committee 1928 to 1955.

In Saint Paul's Cathedral there is a plain tablet erected to the memory of the church's great architect, Sir Christopher Wren, with the simple words: "Lector, si monumentum requiris, circumspice." In the hearts of the members of the Research Study Club there may fittingly be erected such a tablet with such an inscription, honoring him who was in a real sense its founder and architect.

* * *

"Seest thou a man diligent in his business? He shall stand before kings." *Proverbs* 22:29. H.S.M.

IN MEMORIAM

ISAAC HAMPSHUR JONES, M.D.*

Dr. Isaac Hampshur Jones, in his prime and even mature years, would want no tears, no sadness, on this occasion; he would remind us that solace has an old meaning: to make cheerful, to entertain, to amuse. Dr. Jones was a master at giving this kind of solace. He loved life, as he loved truth, beauty, and good cheer. He would have us meet death as Socrates did, with courage and a cheerful countenance.

Dr. Jones would have us recall from the Phaedo that after Socrates had drunk the hemlock, his close friends, Crito, Apollodorus and others, were unable to restrain their tears. Socrates alone retained his calmness. "What is this strange outcry," he said. "I sent away the women mainly in order that they might not offend in this way." . . . and he walked about until, as he said, his legs began to fail. He began to grow cold from the feet upward, and when the coldness reached his groin, he said (and these were his last words): "Crito, I owe a cock to Asclepius; will you remember to pay the debt?"

In this same calm and cheerful manner Dr. Jones moved through life and even into the portal of the hereafter. He probably greeted St. Peter with an amusing and appropriate story. His last words to me, a few months ago, spoken at only a decibel above a whisper, were in story-form. He was a superb story teller; he had an apt one for every occasion; even a telephone conversation with him rewarded you with at least one good story.

Dr. Jones has fashioned mansions and monuments that will long survive him. Even his earthly mansions were built of materials—stone and bronze and oak—to stand a thousand years: but he built even more stately mansions. *Flying Vistas*, a book he wrote in the thirties, as a tribute to the work of his colleague, Dr. Theodore Lyster, whom he credited with the creation of a new specialist, the Flight Surgeon, is a monument to Col. Jones, no less than to Gen. Lyster, for devising tests for screening pilots who *are*, from those who *are not*, fit to fly. The subtitle for *Flying Vistas* is *The Human Being, as Seen Through the Eyes of the Flight Surgeon.* The book treats the seeing, equilibrating and motion-sensing functions of the pilot as indispensable parts of the airplane in safe and effective flight. Chapter III, for example, deals with man's eyes and their role in flying. Note how Isaac Jones, the teacher and writer, begins this chapter. I quote:

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*Eulogy delivered by Vern O. Knudsen, Vice Chancellor and Dean of the Graduate Division University of California, Los Angeles

> All of us old Amherst men sing to this day:
>
> *Here's to 'Tip with dismantled dome—*
> *He never was known to use a comb.*
>
> "'Tip' Tyler taught biology—there was wisdom in that vast bald head. We freshmen soon found out that we had to learn—'Ontogenesis is a brief recapitulation of Phylogenesis.' If you could say that, you passed; if not, you flunked."

More scholarly than *Flying Vistas* are two other books, which are largely the work of Dr. Jones: *The VIII Nerve*, which deals with the physiology and pathology of the internal ear and its associated nervous system, and his earliest book, *Equilibrium and Vertigo.*

Dr. Jones liked to work, and especially to write, with his colleagues. During the ten years that he and I collaborated, he and his co-authors published at least a score of clinical and scientific papers, concerned mostly with impaired hearing—how to diagnose it; and how to correct it. In his later years, his scholarly interests were mainly in vitamins and their applications to otolaryngology. In collaboration with such colleagues as Eugene R. Lewis, Leland Hunnicutt, Jerome A. Hilger and H. S. Muckleston, he prepared annual reviews on vitamins and nutrition. The last one was published in THE LARYNGOSCOPE in December, 1955.

Dr. Jones was a distinguished practicing otologist and organizer as well as a prolific writer for medical journals. He was often consulted by other otologists when they were unsure about their diagnoses or treatments. He organized the Los Angeles Study Club and the Mid-Winter Clinical

Course more than 30 years ago. The latter now attracts more than 300 Eye-Ear-Nose-and-Throat Specialists to Los Angeles each January for a two-week postgraduate course. Many of the world's foremost ophthalmologists and otolaryngologists have lectured and conducted clinics at these annual sessions. Through all the planning and conducting of these sessions the effective guidance of Dr. Jones was evident, but Dr. Jones was modestly in the background.

He and his life-long friend, the late Regent Edward A. Dickson, played a key role in founding the Tennis Patrons' Association, of which Dr. Jones was long the President.

Dr. Jones lived in a home of beauty, culture and refinement, where mutual love between him and his late wife Emilie, and his son Austin, and Austin's wife Fernande and their two children, contributed much to his abundant life. Besides this good life at home, Dr. Jones enjoyed the respect, devotion and affection of a very large circle of friends, colleagues, and patients. He enriched the treasures of his own mind and heart by living and sharing with others, *but he gave more than he received.* He loved almost everything, from the cooking and stories of Pierre, the Basque chef at the Beach Club, to the finest prose and poetry. I close by reading one of his favorite sonnets, written by a U.C.L.A. colleague, the late Professor of Art, George J. Cox. The title is *Survival*:

When from the poles eternal ice sweeps down
To fold now tropic lands in lunar sleep,
And this dead earth, as a new risen moon,
On other planets other tides shall keep;
When the exhausted sun in cindrous gloom
Threads a dark orbit through unlighted stars,
And the slow grind of timeless years consumes
In silent night all trace of senseless wars;
When all the art and science of mankind
Are with the dinosaur alike decayed,
Brave pioneers of better worlds shall find
Unravished loveliness no greeds betrayed—
For of the triumphs of the human mind
Surely the beauty of truth is saved.

THE RESEARCH STUDY CLUB OF LOS ANGELES

Announces its Twenty-Sixth Annual Mid-Winter Clinical Convention in Ophthalmology and Otolaryngology, January 14 through January 25, 1957.

For "Ear-Nose-Throat," the main Guest Lecturers will include Dr. Louis H. Clerf, Dr. George W. Shambaugh, Jr., and Dr. Maurice H. Cottle.

Mrs. Spencer Tracy, Founder and Director of the John Tracy Clinic of Los Angeles for Deaf and Hard-of-Hearing Children and Their Parents, will speak to the "Ear-Nose-Throat" group the opening day of the Convention on "The Role of Parents in the Education of a Deaf Child."

Dr. H. Russell Fisher, a pathologist of note, will bring us up-to-date in his field.

Instruction Courses in Otolaryngology will be provided by three main speakers, Dr. Clerf, Dr. Shambaugh and Dr. Cottle, as well as by Dr. Aram Glorig, Jr., and Douglas E. Wheeler, Ph. D., both of Los Angeles and authorities in Audiometry.

The main Guest Lecturers for "Eye" program will be Dr. John M. McLean and Dr. Graham Clark, both of New York City; and Dr. Samuel J. Kimura of Berkeley, Calif.

Instruction Courses in Ophthalmology will be conducted by Guest Speaker, Dr. Kimura; Dr. Julian N. Dow, Dr. Robert V. Shearer, Dr. Alfred R. Robbins, and a symposium composed of Dr. Clarence H. Albaugh, Dr. John P. Lordon, and Mr. Russell L. Stimson—all of Los Angeles.

The first week will be devoted to the "Ear-Nose-Throat," from Monday to Friday, January 14 through January 18. The "Eye" week will cover Monday through Friday, January 21 through January 25. Those who confine their work to only one of these specialties may complete either subject in one week, or remain for the full two weeks.

As a period of relaxation, Wednesday afternoons will be

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As a period of relaxation, Wednesday afternoons will be

free for sight-seeing, shopping, golf, radio broadcasts, and the enjoyment of points of interest in and about Los Angeles, including the famous Huntington Library with its collection of rare books and art.

Motion pictures of "Ear-Nose-Throat" subjects will be shown during the first week—Monday, Tuesday, Thursday and Friday, 3:00 to 4:00 p.m.

"Eye" subjects will be presented on Wednesday afternoon of the second week for those who prefer to take advantage of this opportunity to view films dealing with subjects in Ophthalmology.

The American Laryngological, Rhinological and Otological Society, Western Section, will hold its Annual Meeting in San Francisco between the two weeks of the Convention on Saturday afternoon, January 19, and Sunday morning, January 20. This will permit members to attend the Triologic meeting and return to Los Angeles for the "Eye" week. Programs of the San Francisco meeting will be available at the registration desk at the Elks Club.

On Monday evening, January 21, the annual banquet of the Los Angeles Society of Ophthalmology and Otolaryngology will honor the Guest Lecturers of the "Eye" week. This will take place at 7:00 p. m. in the El Venado Room of the Elks Club.

The Women's Entertainment Committee will arrange for various events of interest to the wives of visiting members during the Convention. This Committee will appreciate information as to whether your wife will attend with you, which will give the Committee an estimate as to the number of wives for whom to plan entertainment. Details of all such events will be printed and available at the registration desk.

Unless you have your own plans for a place to stay, it is advised that you write for reservations at an early date to Mr. R. B. Philbin, Manager of the Elks Club, 607 South Parkview Street, Los Angeles 57. He will endeavor to arrange suitable quarters for you in a conveniently located hotel, and will confirm the reservation by return mail. The Elks Club is the locale for the entire daily program.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

The meeting dates of the Sixth International Congress of Otolaryngology are again emphasized as May 5th through May 10th, 1957. The scientific program for the Plenary Sessions is now complete and is as follows:

CHRONIC SUPPURATION OF THE TEMPORAL BONE.

OPENERS: Marcus Diamant, Central County Hospital, Halmstad, Sweden—Anatomical Etiological Factors in Chronic Middle Ear Discharge.

Luzius Rüedi, Zurich, Switzerland—Pathogenesis and Treatment of Cholesteatoma in Chronic Suppuration of the Temporal Bone.

Horst Wullstein, Director, Otolaryngological Clinic, University of Würzburg, Germany—Surgical Repair for Improvement of Hearing in Chronic Otitis Media.

DISCUSSERS: A. Tumarkin, Liverpool, England; Juan Manuel Tato, Buenos Aires, Argentina; T. E. Cawthorne, London, England; Fritz Zöllner, Freiburg, Germany.

COLLAGEN DISORDERS OF THE RESPIRATORY TRACT.

OPENERS: Hans Selye, Director, Institute of Experimental Medicine and Surgery, University of Montreal, Faculty of Medicine, Montreal, Canada.

Introduction:

Michele Arslan, Padua, Italy—The Upper Respiratory Tract.

Leslie Gay, Physician-in-Charge, Allergy Clinic, The Johns Hopkins Hospital, Baltimore, U. S. A.—The Lower Respiratory Tract.

DISCUSSERS: Victor E. Negus, London, England; Branimir Gusic, Zagreb, Yugoslavia; Aubrey G. Rawlins, San Francisco, U. S. A.; Henry L. Williams, Rochester, Minn., U. S. A.

PAPILLOMA OF THE LARYNX.

OPENERS: *Jo Ono, Tokyo, Japan—Etiology.*

Plinio de Mattos Barretto, Faculty of Medicine, University of Sao Paulo, Brazil.

Diagnosis:

F. C. W. Capps, London, England—*Therapy.*

DISCUSSERS: C. A. Hamberger, Goteborg, Sweden; Pedro Hernandez Gonzalo, Havana, Cuba; Eelco Huizinga, Groningen, Netherlands; Albert von Riccabona, Vienna, Austria.

Anyone planning to attend the Congress and who has not yet registered should do so immediately in order to obtain hotel registration priority.

For more detailed information pertaining to the Sixth International Congress please communicate with the General Secretary, 700 N. Michigan Ave., Chicago 11, Ill., U. S. A.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. John R. Lindsay, 950 East 59th Street, Chicago 37, Ill. ....
Vice-President: Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.
Secretary-Treasurer: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Statler Hotel, Washington, D. C., May 4, 1957.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. LeRoy A. Schall, Boston, Mass.
First Vice-President: Dr. Henry M. Goodyear, Cincinnati, Ohio.
Second Vice-President: Dr. Robert E. Priest, Minneapolis, Minn.
Secretary: Dr. Harry P. Schenck, Philadelphia, Pa.
Treasurer: Dr. Fred W. Dixon, Cleveland, Ohio.
Meeting: Statler Hotel, Washington, D. C., May 3, 1957.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Percy Ireland, Toronto, Canada.
President-Elect: Dr. Lewis F. Morrison.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Statler Hotel, Washington, D. C., May, 1957.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: John R. Lindsay, M.D., Chicago, Ill.
Vice-Chairman: James W. McLaurin, M.D., Baton Rouge, La.
Secretary: Hugh A. Kuhn, M.D., Hammond, Ind.
Representative to Scientific Exhibit: Walter Heck, M.D., San Francisco, Calif.
Section Delegate: Gordon Harkness, M.D., Davenport, Iowa.
Alternate Delegate: Dean Lierle, M.D., Iowa City, Iowa.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., Oct. 13-19, 1957.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Clarence W. Engler, 2323 Prospect Ave., Cleveland, Ohio.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia, Pa.
Meeting: Mark Hopkins Hotel, San Francisco, Calif., May 21-23, 1958.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill., October 6-12, 1957.

THE AMERICAN RHINOLOGIC SOCIETY.

President Dr Ralph H Riggs, 1513 Line Ave, Shreveport, La
Secretary Dr James Chessen, 1829 High St, Denver, Colo
Annual Clinical Session Illinois Masonic Hospital, Chicago, Illinois, October, 1956
Annual Meeting Palmer House, Chicago, Illinois, October, 1957

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President Dr D M Lierle, University Hospital, Iowa City, Iowa
Secretary Treasurer Dr Michael H Barone 468 Delaware Ave, Buffalo 2, N Y
Meeting Palmer House, Chicago, Ill, October, 1957

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY

President Dr Irvin J Fine 506 New Brunswick Ave Perth Amboy, N J
Secretary Dr William Schwartz, 224 Lexington Ave Passaic, N J
Meetings Quarterly

OTOSCLEROSIS STUDY GROUP

President Dr Gordon D Hoople, 1100 East Genesee St, Syracure, N Y
Secretary Dr Lawrence R Boies, University Hospital, Minneapolis 14, Minn
Meeting Palmer House Chicago Ill October, 1957

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY

President Dr Joseph Gilbert 111 E 61st St, New York, N Y
Vice President Dr Kenneth Hinderer, 402 Medical Arts Bldg, Pittsburgh, Pa
Secretary *Dr Louis Joel Feit, 66 Park Ave, New York 16, N Y*
Treasurer Dr Arnold L Caron, 36 Pleasant St, Worchester, Mass

PAN AMERICAN ASSOCIATION OF OTO RHINO LARYNGOLOGY AND BRONCHO ESOPHAGOLOGY

President Dr Jose Gros Havana, Cuba
Executive Secretary Dr Chevalier L Jackson 3401 N Broad St, Philadelphia 40, Pa., U S A.
Meeting Sixth Pan American Congress of Oto Rhino Laryngology and Broncho Esophagology
Time and Place Brazil, 1958

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

President Dr Arthur W Proetz Beaumont Bldg, St Louis, Mo
General Secretary Dr Paul Holinger, 700 No Michigan Ave, Chicago 11, Ill
Meeting Statler Hotel Washington, D C, May 5 - 10, 1957

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY

President Dr Chevalier L Jackson
Vice-President Dr John J O Keefe
Treasurer Dr Joseph P Atkins
Secretary Dr Louis E Silcox
Historian Dr Herman B Cohen
Executive Committee Dr Harry P Schenck, Dr, Benjamin H Shuster, Dr William A Lell, Dr, William J Hitschler

BALTIMORE NOSE AND THROAT SOCIETY

Chairman Dr Walter E Loch, 1039 No Calvert St, Baltimore, Maryland
Secretary Treasurer Dr Theodore A Schwartz

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

President Dr Raymond Kerwin 310 So Michigan Ave, Chicago, Ill
Vice President Dr Frank Wojniak 6132 So Kedzie Ave, Chicago Ill
Secretary Treasurer Dr Stanton A Friedberg, 122 So Michigan Ave, Chicago, Ill
Meeting First Monday of each Month, October through May

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. J. L. Levine.
Vice-Chairman: Dr. Russell Page.
Secretary: Dr. James J. McFarland.
Treasurer: Dr. Edward M. O'Brien.
Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Frederick A. Holden, Medical Arts Building, Baltimore 1, Maryland.
Chairman-Elect: Dr. Sherman B. Forbes, 706 Franklin Street, Tampa 2, Florida.
Vice-Chairman: Dr. William J. G. Davis, 1632 K Street, N. W., Washington 6, D. C.
Secretary: Dr. G. Slaughter Fitz-Hugh, 104 East Market Street, Charlottesville, Virginia.

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Howard L. Mitchell, Lexington, Va.
President-Elect: Dr. L. Benjamin Sheppard, 301 Medical Arts Building, Richmond 19, Va.
Vice-President: Dr. Edgar Childrey, Jr., Professional Building, Richmond, Va.
Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Va.
Annual Meeting.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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RECRUITMENT TESTS AT LOW SENSATION LEVELS.*

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Testing the loudness recruitment phenomenon is invaluable in routine clinical work. It can materially assist the otologist in arriving at a more definite diagnosis as to the site of pathology than was possible less than ten years ago.[1] In addition to the diagnostic point of view, recruiting ears form a separate class in rehabilitation, because the functional properties of these ears often differ fundamentally from those of ears without recruitment.

Although the recruitment tests proper—Fowler's binaural and Reger's monaural balance—can be performed in a great majority of patients,[2] there remain a few cases where the deafness in both ears and at all frequencies is so similar that neither of the two procedures can be employed satisfactorily. It is in this comparatively rare connection that the need for a good substitute for the balance test arises.

Certain special features of some recruiting ears are frequently elaborated upon and presented as being characteristic of the whole group; it is regrettable, however, that most of the original studies have not been fully validated against the classical loudness balance tests and have proved disappointing in critical control experiments.

One of the characteristics thought by some investigators to

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label all cases of recruiting deafness, is a less than normal intensity difference limen (IDL). The inconsistencies of this reasoning were sufficiently stressed earlier[2] and there is no need to repeat here the arguments against any close relation between recruitment and various IDL tests. Control experiments have shown unquestionably that at least for the present there are no such IDL tests as might be substituted for the proper loudness balance measurement.[2,3,4,5]

Hallpike and Hood, and their group have repeatedly stressed the occurrence of another characteristic of recruiting ears; *viz.*, the development of pathological per-stimulatory adaptation (also called per-stimulatory fatigue.[6,7,8,9] In their experiences a recruiting ear cannot sustain the initially perceived loudness (at sufficiently high intensity levels) and fails in performance. During continuous three-minute stimulations a clear decrease in loudness follows; the initial difference at threshold may again appear also at louder levels between the ears, or the decrease may even result in the reversal of loudness recruitment. In other words, the ear fails so much that the initial threshold difference increases at high sensation levels.

Dix and Hood,[7] and Hood,[9] have carried the weight of these findings into clinical application. Because, as they think, a small IDL is a corollary phenomenon with loudness recruitment, the size of the IDL must also follow the fluctuations perceived in the loudness of sustained tones in a recruiting ear. This was exemplified with threshold tracings in two cases of Ménière's disease; automatic recording showing excursions of the width of less than 5 db during the first minute, while after two or three minutes' stimulation they increased to as much as 15 db. In addition, the pathological adaptation manifested itself by a decrease in sensitivity; after three minutes' testing the threshold was about 15 db worse than at the beginning of the test.

On the basis of these findings Dix and Hood[7] doubted the validity of the results of those authors who had earlier opposed the use of IDL tests. The main argument against Lund-Iwersen[4] and against Liden and Nilsson[3] was that they used sustained tones while measuring recruitment or IDL, and, therefore, their results could not be considered reliable.

It is quite true that to demonstrate loudness recruitment, the proper procedure is to use interrupted stimuli alternately in the ears, preferably with a short interval between the pair of tones. The point of the two articles, however, was not so much in recruitment testing but in the fact that the size of the IDL was very much the same in all different groups of deafness, or in normals. Had the test results of these studies been faulty, as Dix and Hood[7] imply, then they would have been manifested in larger than normal IDLs for recruiting cases owing to sustained stimulation and pathological adaptation; however, this was not the case.

In a discussion of Hood's[9] paper in 1953, I[10] pointed out some of the inconsistencies in the assumed close relation between a small IDL and loudness recruitment on theoretical grounds, and by reviewing some of the contradictory studies published on the subject. It was concluded that while a small IDL may often be true in recruitment, it does not necessarily mean that it would be otherwise in any other group of deafness, and that a fairly large IDL can also appear in recruiting cases.

It was also shown later[11] that the occurrence of pathological adaptation is not so common a phenomenon in recruiting deafness as to jeopardize all tests that have been made with sustained tones. In a series of 51 recruiting ears only 14 per cent showed as large decreases at the loudness levels as would be needed to abolish the recruitment phenomenon and to differentiate them from normal ears.

While dealing with pathological adaptation at threshold level, Dix and Hood[7] stated that diminished sensitivity during sustained stimulation occurs also in a variety of other perceptive disorders, including lesions of the VIIIth nerve. Although they considered the physiological basis of this phenomenon to be different in these cases, they concluded that a loss in the threshold sensitivity is by no means an indication of the presence or absence of loudness recruitment.

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In a later paper Hood[12] regarded this test as valuable in the clinical differentiation of end-organ disease. The amount of sensitivity loss during a modified threshold stimulation was from 20 to 30 db during only 70 seconds stimulation. The possible occurrence of this phenomenon in nonrecruiting perceptive deafness was not dealt with.

Decreased sensitivity during continuous threshold stimulation has been noted also by other investigators. It was reported in three patients by Reger and Kos[13] in 1952: the amount of the shift varied from 10 to 30 db. Two of the cases were recruiting, the third a verified case of acoustic tumor. In the latter case the sensitivity loss was most marked.

These observers pointed out, however, that all individuals with recruitment did not show temporary threshold shift, nor did it occur in every case of nonrecruiting perceptive deafness. Recently Kos[14] again reported similar threshold shifts in a case of VIIIth nerve atrophy and in a case of pinealoma, confirmed by surgery.

In their latest papers Hood[12] and Kos[14] seem to hold slightly diverging opinions regarding the occurrence of threshold sensitivity loss. Both have earlier expressed their view of this phenomenon occurring in both end-organ and nerve fiber deafness.[7,13] Hood now speaks of it as one characteristic of end-organ deafness while Kos supports mainly the opposite view.

Dix and Hood,[7] Hood,[9] and Reger and Kos[13] have all implied that a small IDL, diminished excursions in the threshold audiogram and recruitment are equivalent. In fact the two first mentioned phenomena are considered to be measures of the IDL, and this term is used interchangeably for either measure. It is probable that small threshold variability and recruitment go parallel, but not, as stated earlier,[2,15] because a Békésy-type audiogram is a measure of a true IDL but rather because it is a measure of the variability around the patient's threshold. Small variability indicates that the audibility goes up rapidly; large variability is an indication of a clearly slower relation between the two. It must be stressed here again that it furnishes us with the knowledge of this relation only at the threshold level; in fact, according to the observations of Harris, Haines and Myers[16] on various types of recruitment function, one is quite uncertain when evaluating the presence or absence of recruitment from the Békésy-type curve alone. True, one might not be much misguided in cases where the threshold excursions are small, but might fail in many cases with normal excursions but complete recruitment at louder levels.

Earlier, when measuring pathological adaptation at above threshold levels, the writer[11] also made some preliminary records of sustained tone threshold recordings during four minutes' stimulation. In this paper I supplemented these findings on the basis of a greater number of cases. An attempt is also made to study the relation between the recruitment phenomenon and the width of the excursions measured with a self-recording audiometer to reveal the possible advantages and limitations of the latter method in expressing the presence of recruitment.

TESTING PROCEDURE AND APPARATUS.

Air and bone conduction thresholds were measured at frequencies one or one-half octaves apart, employing a Peters SPD-2 audiometer. The threshold was determined with the method of limits using interrupted, about 1 to 2 seconds stimuli. The PDR-10 earphones had reasonably flat response characteristics.

Recruitment testing was done by applying tones of 1.5 seconds duration with an interval of 0.5 seconds alternately to the ears. Either Fowler's binaural or Reger's monaural technique was used. A frequency was selected at which the other ear was normal, or in Reger's balance, a neighboring frequency in the same ear was normal.

If both ears showed decreased sensitivity but in unequal quantities, the balancing procedure was nevertheless employed. The smallest acceptable difference between the ears, or frequencies, was 20 db, the highest 55 db. These differences apply also in cases where the other ear was normal; with this method the danger of shadow hearing was eliminated.

In both procedures a balance was obtained at settings of 20, 40, 60, 80 and 100 db above the threshold of the better ear. The highest intensity obtainable was 100 db above zero, corresponding to 116 db re 0.0002 microbar.

At each balancing level at least three different judgments of equal loudness were obtained. The equality was approached both from "above" and "below,"[2] and the patient was allowed to listen to as many pairs of tones as he wished before giving the judgment of "equal" or "different." A case was labeled recruiting only if the difference at threshold disappeared completely at louder levels.

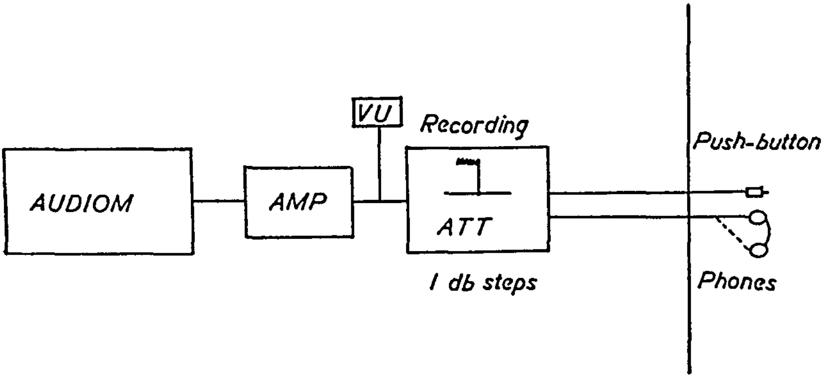

Fig. 1. Block diagram of apparatus.

The threshold audiometry using sustained tones was done with the method of adjustment. The Peters SPD-2 audiometer was used as a separate sound source to feed the tones to the first channel of the Bekésy-type audiometer. The intensity of the tones was controlled before the attenuators with a Pegel voltmeter. The block diagram of the apparatus is shown in Fig. 1.

The listener controlled the intensity of the tones in his ear by means of a push-button which regulated the intensity in one db steps at a rate of 140 db per minute and operated the instantly reversible motor moving the audiogram chart. The subject was instructed to push the button every time he heard a tone, and release it as soon as the tones disappeared. The recording pen traced the excursions automatically in the adjacent room.

The width of the excursions demonstrates directly the amount of variability around the threshold as a function of time. The changes in sensitivity are manifested by either slowly ascending or descending threshold curves, and if there is no change, the tracing pursues a steady course.

In certain cases supplementary speech tests were made. The technique followed the principles described earlier.[17]

Finally, when it was necessary, thermal noise was delivered through the second channel of the Békésy-type audiometer for masking purposes. The principles involved and exact intensities used are adequately described elsewhere.[18]

MATERIAL.

The great majority of the persons on whose records this report is based are patients in my private practice. They represent all the various groups of deafness, although attention is focused mainly on patients with perceptive deafness. Unfortunately, cases with nonrecruiting perceptive deafness are few in number, and the material includes no patients with acoustic tumors; on the other hand, recruiting perceptive deafness is sufficiently represented.

Much attention is given to good patient-tester relationship in the Audiology Laboratory. If the subject does not fully understand his share in the test, there is little hope for accuracy in the results. The special tests were explained as simply and comprehensively as possible.

The familiarity of the patient with auditory testing may give him an advantage over a person undergoing both the ordinary threshold tests and supplementary special tests for the first time. This point was kept in mind while analyzing the results.

The conductive group consisted of six persons. Two were under treatment for subchronic otitis media, two were unilateral cases of adhesive otitis, and two were patients with otosclerosis of rather short duration; their hearing loss being still mainly unilateral. All showed good bone conduction and normal intelligibility for speech.

The nonrecruiting perceptive group includes two persons with presbycusis and with a prominent high tone loss: one of them (O. E.) also had a superimposed conductive lesion. In four cases of partial VIIIth nerve degeneration the etiology could not be clearly defined.

In the recruiting perceptive group 27 cases were studied. The diagnosis was Ménière's disease in 10 cases, noise trauma in two cases, and in 15 cases the etiology could not be definitely determined.

RESULTS.

If a person with normal hearing makes a threshold tracing with a self-recording apparatus, the tracing pursues a steady course indefinitely;[13] however, small fluctuations in this course (of the order of ±5 db) should be considered normal. This amount of change can easily be caused by slight inattention while listening, while shifting position or breathing deeply, or it may arise if the noise level of the room undergoes small variations. Using the method of limits, ±5 db is also generally considered to represent the experimental error.

All the individual results are presented in Table I. The threshold at the tested frequency, the amount of temporary threshold shift after two, three and four minutes of sustained stimulation, the width of the excursions during the first minute of recording, the amount of change in the amplitude of the excursions during stimulation, and finally the diagnosis, appear in the table.

The reference intensity for the temporary threshold shift is the midpoint of the excursions obtained during the first half minute of stimulation. In some cases, in which the initial trend was not clear, the midpoint of the tracings obtained during the first minute of stimulation was used.

In the conductive cases the threshold tracings lay on both sides of the reference level, and none of the values deviated from it by more than 5 db. It seems that the threshold remained stable, and the ears did not become less sensitive during sustained stimulation.

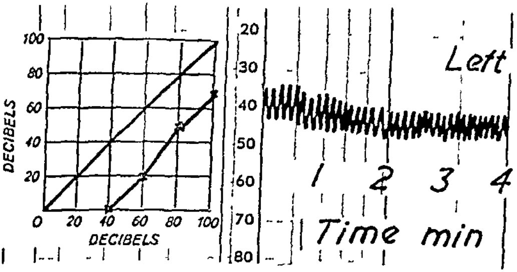

Fig 2 Case A M pharmacist, age 37 years The patient has adhesive otitis in the left ear The audiograms showed normal hearing in the right ear and a moderate 30-40 db conductive hearing loss in the left ear.

The binaural loudness balance test at 500 cps shows no recruitment. The four minutes threshold tracing at the same frequency shows a normal course with some reduction in the width of the excursions during the last two minutes

The width of the excursions during the first minute of stimulation varied somewhat from individual to individual. It seems fair to say that generally they averaged 7 to 8 db, the lower limit being around 5 db and the upper below 15.

The width of the excursions did not change much during stimulation. The change was of the order of ±1-2 db, except in one case with the largest initial variability (A. M.). Here the final tracing was 7 to 8 db; there was not so much change in the lower limit of the tracing, but the upper limit (the-just-not-heard-tone) decreased relatively much more.

All these patients had had audiometric tests made earlier. Fig. 2 shows the results in one case (A. M.).

In the nonrecruiting perceptive group the initial width of

TABLE I.

INDIVIDUAL RESULTS.

Case	Threshold db	Frequency cps.	Threshold Shift db 2 Min.	Threshold Shift db 3 Min.	Threshold Shift db 4 Min.	Excursions Initial Width db	Excursions Change During Stimulation db	Diagnosis
H. L.	40	500	0	— 1	— 1	7- 8	Larger by 1 db	Conductive deafness; no recruitment
S. L.	30	1000 dx	+3	+ 4	+ 3	6- 8	Larger by 1-2 db	
	50	1000 sin	—2	— 2	— 2	7- 8	No change	
T. S.	10	1000 dx	—1	— 1	— 1	5- 7	No change	
	45	1000 sin	—2	+ 4	+ 5	5- 6	Larger by 1-2 db	
A. M.	40	500	+4	+ 4	+ 4	8-12	Smaller by 3-5 db	
A. A.	30	1000	+2	0	0	7- 8	Smaller by 1-2 db	
T. P.	20	1000	+2	0	— 1	6-10	No change	
T. H.	40	1000 sin	—3	— 4	— 4	7- 8	Smaller by 2-3 db	Perceptive deafness; no recruitment
	60	1000 dx	—2	— 4	— 6	7- 8	Smaller by 2-3 db	
M. L.	15	1000 sin	—1	+ 1	0	6- 8	No change	
	40	1000 dx	—1	+ 3	+ 4	6- 8	Smaller by 1-2 db	
E. L.	40	6000 sin	—2	+ 4	+ 3	6- 8	Larger by 2-3 db	
	50	6000 dx	+2	+ 7	+10	10-15	Larger by 2-3 db	
A. W.	20	250	—1	— 2	+ 1	10-16	Smaller by 3-4 db	
O. E.	50	2000	+2	+ 5	+ 7	7- 9	Smaller by 2-3 db	
K. K.	15	2000	—1	+ 1	0	3- 4	No change	Perceptive deafness with asymptotic recruitment
	40	4000	+1	+ 1	+ 1	3- 4	No change	
U. M.	35	4000 sin	+9	+20	+27	1- 3	Smaller by 1 db	
	65	4000 dx	+7	+ 7	+ 6	1- 2	No change	
M. M.	45	1500 sin	+3	+ 4	+ 3	2- 3	No change	
	70	1500 dx	+3	+ 2	+ 3	2- 3	No change	
T. S.	20	250 dx	+4	+ 6	+ 6	7-10	Smaller by 1-2 db	
	40	250 sin	+1	+ 1	0	6- 8	Larger by 1-2 db	
P. L.	75	3000	0	0	+ 1	3- 5	No change	
M. V.	40	6000	+3	+ 5	+ 7	2- 3	Smaller by 1 db	
A. P.	30	4000	—4	— 2	— 1	3- 4	Smaller by 1-2 db	

TABLE I—Continued

Case	Threshold db	Frequency cps	Threshold Shift db			Excursions		Diagnosis
			2 Min	3 Min	4 Min	Initial Width db	Change During Stimulation db	
V K	75	500	+5	+ 5	+ 4	10 11	No change	
	70	1000	+2	+ 2	+ 2	5 7	Smaller by 3 db	
	55	2000	+7	+ 6	+ 6	5 7	No change	
	60	4000	+2	+ 4	+ 3	5 8	Smaller by 4 db	
V U	50	4000 dx	+2	+ 5	+ 3	8 10	Smaller by 4 5 db	Perceptive deafness with asymptotic recruitment
	30	4000 sin	+2	+ 3	+ 2	3 5	No change	
	15	2000 sin	0	+ 1	+ 2	7 10	No change	
S A	65	1000	+4	+ 4	+ 4	3 5	No change	
T L	30	4000	—2	— 1	+ 2	4 5	No change	
E U	45	4000	0	+ 1	0	2 3	No change	
P L	45	4000	—1	— 3	— 4	2 3	No change	
U A	70	4000	+4	+ 4	+ 6	4 7	No change	
U L	30	500	+1	— 1	— 1	7 10	No change	
V U	60	1000	+2	+ 1	— 3	8 10	No change	
E H	20	4000 dx	—3	— 6	— 5	7 10	Smaller by 4 6 db	Perceptive deafness, with straight line recruitment
T S	50	4000 sin	—2	— 4	— 3	2 3	No change	
T S	45	250	—1	+ 3	+ 3	6 9	No change	
J S	35	500	—1	+ 1	+ 1	7 8	No change	
K N	65	3000	0	+ 1	+ 1	9 12	No change	
S L	60	1000	0	0	+ 5	10 12	Larger by 4 6 db	
J S	30	500	—1	— 1	— 1	5 7	No change	
V U	50	1000	—4	— 5	— 5	8 10	No change	
K K	25	250	—2	+ 1	+ 2	8 9	No change	Perceptive deafness, with delayed recruitment
V V	50	500	0	+ 2	+ 4	5 8	No change	
Y A	30	500	+3	+ 2	+ 2	13 16	No change	
K V	30	500	—2	— 4	— 5	18 20	No change	
	45	1000	+3	+ 3	+ 2	15 18	No change	
	35	2000	+5	+ 6	+ 8	15 16	No change	
	45	4000	+3	+ 6	+ 8	10 16	No change	

the excursions was about the same as in the conductive group, viz., on an average 7 to 8 db. In this group, too, the case showing largest variability (A. W.) at the beginning, exhibited a clear decrease during the test. Except in one case (E. L.), which is shown in Fig. 3, the amplitude of the tracings generally decreased by a few decibels during sustained stimulation.

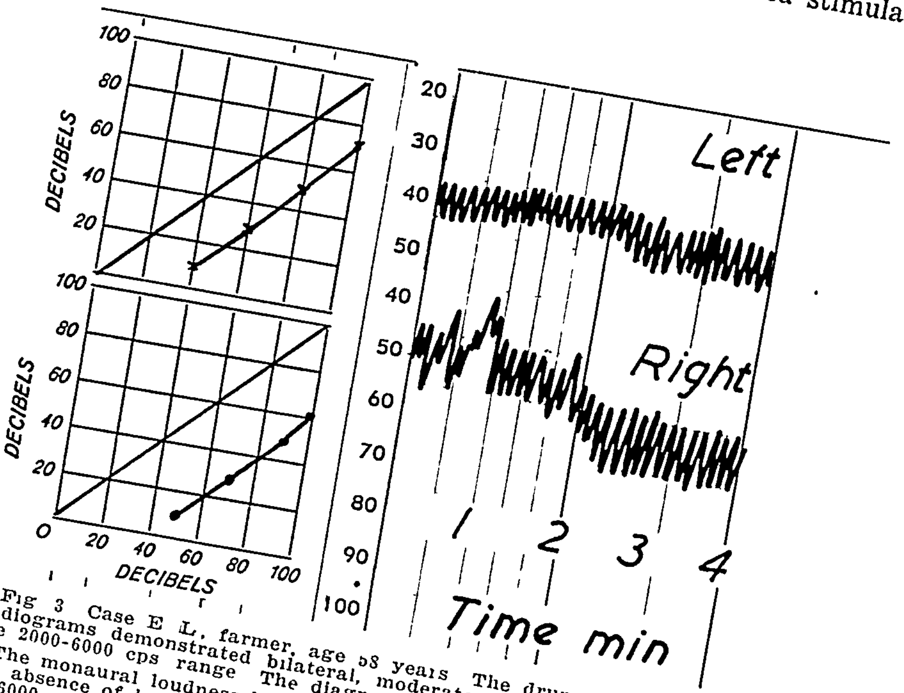

Fig. 3. Case E. L., farmer, age 58 years. The drums are normal. The audiograms demonstrated bilateral, moderate perceptive hearing loss in the 2000-6000 cps range. The diagnosis was presbycusis.

The monaural loudness balance test between 3000 and 6000 cps reveals the absence of loudness recruitment in both ears. The threshold tracings at 6000 cps show a normal course in the left and a 10 db per-stimulatory loss in the right ear during sustained stimulation. The width of the excursions increases about 1 to 3 db in size during stimulation.

The deviations from the initial threshold value during continuous stimulation are not so clear cut. At two frequencies there was no significant change, but in one ear the threshold improved by 6 db and in three ears it dropped 6 to 10 db below the reference level. Because of the small number of cases it is not possible to make generalizations, but it seems obvious that some nonrecruiting perceptively deaf ears may exhibit significant temporary threshold losses during sustained stimulation.

Two of the patients in this group had had audiometric studies made earlier. This did not seem to affect the behavior of the group in any way.

Recruiting perceptive deafness was further subdivided into three different categories on the basis of the recruitment functions. The first group includes all ears where the rise in the loudness function was asymptotic, in the second group are the cases where the line connecting various equal loudness points was a straight one; in the third group the recruitment was delayed at low intensities. This is in line with the various recruitment curves presented earlier by Harris, Maines and Myers.[16]

The group of asymptotic recruitment includes 14 persons, for whom the results are presented at 23 frequencies. At six frequencies the per-stimulatory threshold loss exceeded 5 db. In one case the loss attained the really high value of 27 db after four minutes' stimulation. In all others the tracing was within ±5 db limits from the reference level; the average response, therefore, seems to be similar to that in a normal ear. The audiometric results in the exceptional case (U. M.) are presented in Fig. 4.

The straight line recruitment group consists of the results obtained for eight persons at nine frequencies. The per-stimulatory threshold shift was within normal limits in all these cases.

Delayed recruitment was demonstrated in five cases and the results are shown at nine frequencies. It appears that in one case (at two frequencies) the per-stimulatory threshold loss amounted to 8 db.

The width of the excursions during the first minute's stimulation showed significant individual variations even in the group of asymptotic recruitment. At 15 frequencies it varied from 1 to 5 db and at eight frequencies it varied from 5 to 12 db. It is apparent that the latter values cannot be used as criteria indicating the presence of recruitment, because the same width of excursions occur also in the normal, conductive, and nonrecruiting perceptive group. About two-thirds of the frequencies tested thus agree with the recruitment tests proper.

The nine frequencies tested in the straight line recruitment group include only one where the width of the tracing was as small as 2 or 3 db. In all others the excursions were as wide as in any other nonrecruiting group.

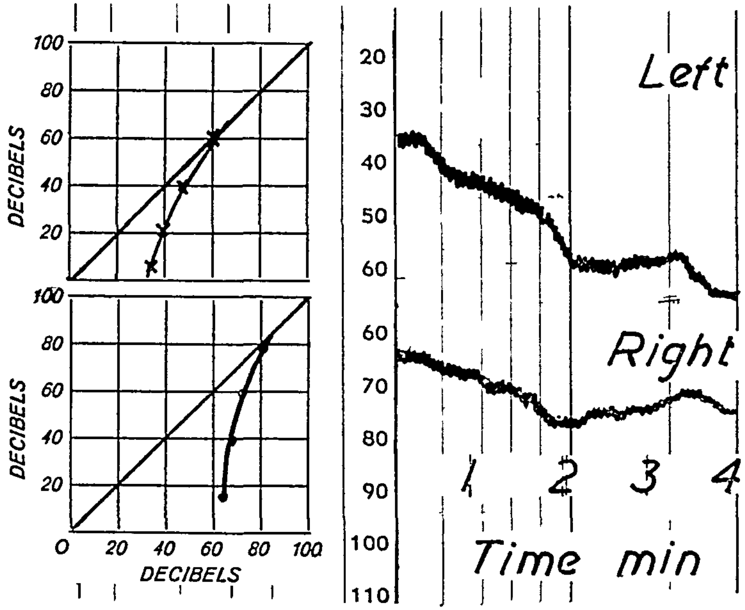

Fig. 4. Case U. M., business executive, age 36 years. The drums are normal. The audiograms revealed bilateral good hearing up to 3000 cps. and severe perceptive hearing loss in the higher range.

The monaural loudness balance between 3000 and 4000 cps. in both ears indicates asymptotic recruitment. The threshold tracings at 4000 cps. demonstrate a marked temporary threshold loss which is exceptionally large in the left ear. The width of the excursions is small and consistent with a recruiting deafness. It may be noted that the amplitude of the excursions decreases somewhat as a function of time.

Finally, at all nine frequencies where recruitment was initially delayed, all threshold tracings were larger than 5 db; therefore, on the basis of the threshold tests alone, one would not place them into the group of recruiting deafness.

The amplitude of the tracings remained stable on an average during four minutes' stimulation. In four cases there was a noticeable decrease (3 to 6 db) which placed the final width below the critical figure of 5 db, and these cases can thus be added to those in which conclusions regarding the presence of recruitment can be made from the threshold studies. Three of them were in the asymptotic recruitment group, one in the straight line recruitment group. It may be noted that in the ordinary Békésy audiogram the excursions were greater than 5 db, corresponding to the initial width of the tracings with sustained one-frequency stimulation.

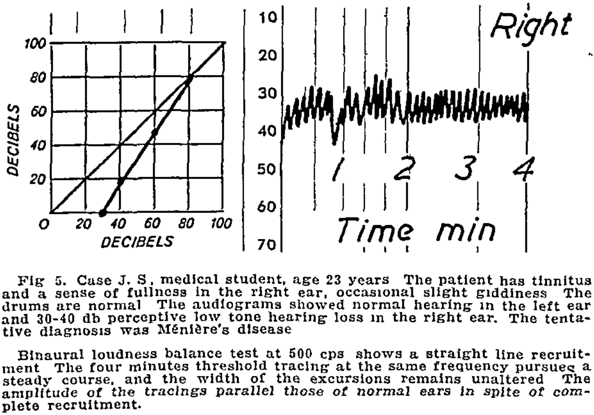

Fig 5. Case J. S, medical student, age 23 years The patient has tinnitus and a sense of fullness in the right ear, occasional slight giddiness The drums are normal The audiograms showed normal hearing in the left ear and 30-40 db perceptive low tone hearing loss in the right ear. The tentative diagnosis was Ménière's disease

Binaural loudness balance test at 500 cps shows a straight line recruitment The four minutes threshold tracing at the same frequency pursues a steady course, and the width of the excursions remains unaltered The amplitude *of the tracings parallel those of normal ears in spite of complete* recruitment.

In only one case the tracings increased in size as a function of time. This case belonged to the group of straight line recruitment (S. L.) and the excursions were even primarily quite large, 10 to 12 db. In a per-stimulatory 80 db sensation level (above the threshold of the normal ear) balance test,[11] with a continuous tone in the impaired ear and an adjustable tone in the good ear, a five minutes' sustained stimulation presented a tracing pursuing a steady course.

In Figs. 5 and 6 are presented two cases as examples of the tests in the straight line and delayed recruitment groups.

About one-fifth of the recruiting patients had been previously tested audiometrically, and they all showed steady threshold excursions. Those with a clear decrease or increase in the width of the excursions were being tested by audiometry for the first time.

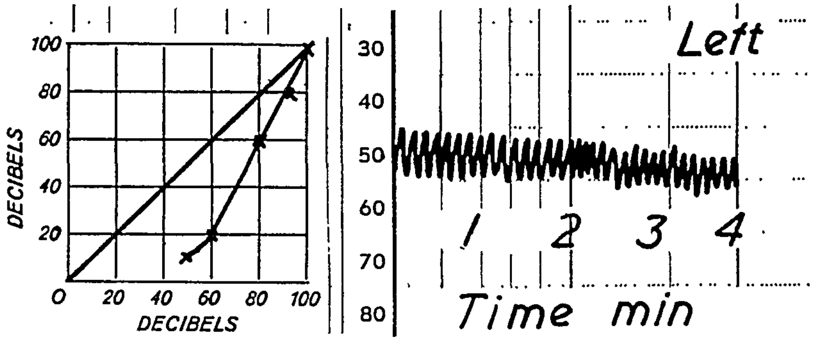

Fig. 6. Case V. V., industrial worker, age 54 years. The patient has had poor hearing in the left ear since infancy. The drums are normal. The audiograms revealed a nearly normal hearing in the right ear, and a marked, about 50-70 db perceptive, hearing loss in the left ear.

Binaural loudness balance test at 500 cps. shows delayed-straight line recruitment. The threshold tracing at the same frequency shows an equal, steady course and the width of the excursions remains unaltered. The threshold tests alone, without proper loudness balance, do not show that there is complete recruitment.

In addition, three patients, whose records are not included in this material, showed recruitment in the balance tests, though the threshold excursions remained between 20 and 30 db. An attempt was made during the following day to accustom them to the self recording technique, but this was unsuccessful. It is apparent that some slowly responding people will not give their true results in this type of test.

Occasionally one also finds some normally hearing and quite intelligent persons who do not succeed in the automatic threshold recording. In spite of repeated attempts the threshold variability remains between 15 and 30 db. On the other hand, there may be normally hearing subjects who present extremely small threshold excursions. One was included in the material used for another study;[19] the excursions were at all frequencies clearly below 5 db, and using interrupted tones, between 1 to 3 db through the whole range from 125 to 4000 cps.

DISCUSSION.

Because it is probable that per-stimulatory threshold shift in conductively deaf persons equals that in normals, only a few cases of this type of hearing loss were included. It seems that in both normal subjects and in those with conductive deafness there are frequently variations of around ±5 db, which must be considered to represent the experimental error. The width of the excursions generally varies between 5 and 15 db, and only minor variations in this amplitude are likely to occur.

In nonrecruiting perceptive deafness there may be cases which show per-stimulatory threshold losses, in this study of the order of 10 db; however, a sufficient number of cases was not available for study, and later work may show some deviations from the above figure. For example, there were no cases of acoustic tumor, and in such ears in particular, Kos[14] has demonstrated quite large threshold losses during sustained stimulation.

The width of the excursions may vary considerably, from 5 to 20 db. Small variations as a function of time can be demonstrated in either direction during sustained stimulation.

In the ears showing recruitment it is possible to obtain really large threshold losses during continuous stimulation. The results in the case presented in Fig. 4 are in agreement with those reported by Hood,[12] but it must be stressed that this was an exceptional case. In seven instances the threshold became worse by 6 to 10 db, and in the large majority of cases the threshold tracing followed the same steady course as in the normal or conductive group.

It must be stated, therefore, that although some large losses may occur the average response will be a steady tracing, or a tracing showing only minor evidence of fatigability. This is in accordance with previously published studies on above-threshold per-stimulatory adaptation tests,[11] and it demonstrates that, at threshold intensities, this phenomenon cannot be used as a differential diagnostic auditory test. Even if Kos'[11] report on auditory nerve fiber degeneration associated with large temporary threshold shift appears to be consistent with later control studies, the fact that extensive fatigability can appear in recruiting deafness, eliminates this test from differential diagnosis.

The division of the recruiting cases into various sub-groups was necessary to test the claim made earlier.[2,15] *viz.*, that the Békésy audiogram does not measure the IDL, but rather the variability around the threshold. Small variability indicates that the audibility increases rapidly with increasing intensity, larger variability is associated with normal or slower than normal function between the two.

We[2] stated that one would expect audibility to be intimately related to loudness. In the above groups, therefore, the smallest variability should occur in the asymptotic recruitment group. The straight line recruitment group may show normal, or less than normal variability, depending to some extent upon the threshold loss and upon the steepness of the recruitment curve. In the group of delayed recruitment the threshold tracings should parallel those of normal subjects.

It may be said that the same assumptions can also be made regarding the size of the IDL in these various groups, and that this neglected recruitment function is the cause of the greatly conflicting results of various IDL studies; however, before such an assumption is made, it must be shown that the number of discriminable steps is the same in a normal ear as in an ear with, for example, a 50 db threshold loss. The burden of getting this evidence is rather on the side advocating the use of IDL on the basis of that assumption.

It seems very hard to understand how a damaged end-organ could be capable of better intensity discrimination than a normal ear with all its delicate, unimpaired structures. It might well be that the discriminable steps in recruiting ears are as large, or larger than in normal ears and fewer in number. That would result in normal, or above normal IDLs; however, if one should measure the IDL at the junction of this large step-wise change, one may get a very small intensity reading.

The results show that in most cases of asymptotic recruitment with rapid growth in audibility, the threshold variability is less than 5 db and indicates recruitment. Even in this group there are some cases in which the excursions are more than 5 db and parallel those in the other diagnostic categories. If the recruitment function is of the straight line type, more cases show normal threshold variability, and in the group of delayed recruitment practically all cases show normal excursions, between 5 and 15 db.

It seems clear that Békésy-type audiometry can never replace ordinary audiometry combined with Fowler's or Reger's balance tests. One is probably not misled in cases where small threshold variability is interpreted as to mean recruitment, but one misses equally many cases showing recruitment in the proper tests but failing to show it in the form of small threshold variability.

Of interest is also the variation in the width of the excursions as a function of frequency in recruiting deafness. This is apparent from the results in this paper, but it seems on the basis of a considerably larger material examined in this clinic, that the variability clearly decreases as the pitch increases. Thus, small threshold excursions are most readily demonstrated at 4000 or 6000 cps. Values indicating recruitment can also be quite easily obtained at 2000 or 1000 cps., but rarely if the frequency is further lowered; in fact, using the type of test procedure described in this paper, I have not yet found a single case showing 1 to 3 db threshold variability at 125 or 250 cps. If the threshold loss is considerable, one may get below 5 db at 500 cps., but in my experience even this is an exception rather than a rule.

This relation further weighs against the theoretical soundness of the IDL tests. If the width of the excursions depended solely on the diminished IDL, here would be an ideal area in which to demonstrate it. Even in the normal ear loudness increases for the low tones faster than at the middle frequencies, and combined with low tone recruiting deafness, the rate should be quite fast and correspondingly the IDL very small. Experimental evidence, however, does not support this view.

The change in the width of the threshold excursions as a function of time is comparable to normal in the majority of recruiting cases. If there is a change during four minutes' stimulation, the variability is more likely to decrease than to increase. The reduction can be large enough to cause the case to be properly placed into the group of recruiting deafness although the ordinary Békésy audiogram, or the first one-minute single frequency tracing indicates greater than 5 db threshold variability. Hood's[9] claim that the threshold excursions increase in size as a function of time in sustained stimulation, indicating the reversal of recruitment and a change-over from small to large IDL values, is not supported, and if there is any change it is generally a decrease.

Another major point should be stressed. It is apparent, even on the basis of the various recruitment functions, that it is quite a hopeless task to invent new tests with the object of revealing the presence or absence of recruitment at or close to threshold. This applies especially to the IDL tests, regardless of what one thinks of the soundness of the idea. It applies equally to Békésy audiometry, which as a rule seems to be reliable in cases where it indicates recruitment. If we want to invent new audiometric tests which reveal the presence of recruitment in all cases (gives the same information as Fowler's or Reger's balance test), then it is necessary to work at those intensities where even delayed recruitment becomes apparent. This means something like 60 to 80 db sensation level (re normal threshold), depending somewhat on the frequency and the threshold loss. Around this level practically all cases will show to which group they belong.

SUMMARY.

A short review is given of diagnostic recruitment tests at low sensation levels, *viz.*, IDL tests, per-stimulatory threshold fatigability and automatic threshold recording technique.

Data are reported on 39 patients belonging to various diagnostic categories. On the basis of the loudness balance functions the cases of recruiting perceptive deafness are further subdivided into asymptotic, straight line, and delayed recruitment groups All cases were tested with alternate binaural judgment at the same frequency or monaural judgment between two frequncies Automatic threshold recording was done with a Bekesy-type audiometer providing intensity changes in 1 db steps at a rate of 140 db per minute

The width of the threshold excursions was generally between 5 and 15 db in normal hearing persons Exceptional cases can be found which exceed both these limits The same applies to the conductive and nonrecruiting perceptive deafness. The majority of cases with asymptotic recruitment showed excursion amplitudes of less than 3 db Most cases in the straight line recruitment group presented larger than 5 db excursions and practically all cases with delayed recruitment showed normal variability.

A per-stimulatory threshold shift of ±5 db is considered normal This also occurred in conductive deafness, while in the non-recruiting and recruiting perceptive deafness some ears showed a loss of the order of 10 db, and in some exceptions, more. It does not seem possible to use this test for subdividing the group of perceptive deafness

The width of the excursions during sustained stimulation can undergo small variations as a function of time in each group The majority show unaltered excursions, and if there is any change, it is likely to be a decrease from the initial values

Finally, it is pointed out that any low sensation level test designed to substitute the loudness balance tests, is likely to fail in many cases. It is necessary to work on higher sensation levels if successful results are expected in all cases

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THE PROBLEM OF POST-TONSILLECTOMY POLIOMYELITIS.*

An Analysis of 888 Cases of Poliomyelitis and 2,813 Tonsillectomies From a District in the Southwest United States, 1949-1953.

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I. INTRODUCTION.

The possible causal relationship between tonsillectomy and poliomyelitis that develops shortly after operation has been recognized since 1910, but did not become a matter of general concern for another 20 years. In addition to this basic consideration, two other issues also began to be discussed as time passed—whether tonsillectomy predisposed not only to poliomyelitis but also to the more severe forms, and whether the removal of the tonsils at any period of life rendered the patient more susceptible to this disease.

The discussion has often, unfortunately, been attended by more heat than light. Experimental studies have not been conclusive. Statistical studies have been equally inconclusive. Sweeping conclusions have been drawn from isolated cases, or from series of cases too small to be of statistical significance. Indeed, a survey of the poliomyelitis-tonsillectomy literature permits one to believe, as he may wish:

1. That removal of the tonsils and adenoids in the so-called "polio season" multiplies the chance of contracting the disease, particularly in its more serious forms; or

2. That the development of poliomyelitis after tonsillectomy can be explained as merely fortuitous; or

3. That it can be explained as the result of exposure before operation.

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A survey of the literature also permits one to believe, as he chooses:

1. That removal of the tonsils increases the susceptibility to poliomyelitis, with the susceptibility extending over many years, if not for life; or

2. That the loss of these structures no more influences the chances of developing poliomyelitis than does the loss of the appendix.

Generally speaking, the literature of post-tonsillectomy poliomyelitis falls into three somewhat overlapping periods:

1. In the first period, running from 1910 to approximately 1930, only isolated cases or small groups of cases were reported. The first really extensive study was by Aycock and Luther,[1] in 1929. During this period the possible relationship between tonsillectomy and poliomyelitis was noted, but the discussion was usually tentative.

2. In the second period, which runs to approximately 1945 *or 1946, much larger series of cases were reported, and the* relationship was regarded as important.

3. In the third period, running from 1946 to the present time, a change in methods of reporting became evident. Observers began to report larger series of cases of poliomyelitis, some of which followed tonsillectomy. They also reversed the emphasis and showed the other side of the picture; that is, they began to record large series of tonsillectomies, some few of which were followed by poliomyelitis. During this period, experienced statisticians also began to analyze the records by sound statistical methods.

3. In the third period, running from 1946 to the present time, a change in methods of reporting became evident. Observers began to report larger series of cases of poliomyelitis, some of which followed tonsillectomy. They also reversed the emphasis and showed the other side of the picture; that is, they began to record large series of tonsillectomies, some few of which were followed by poliomyelitis. During this period, experienced statisticians also began to analyze the records by sound statistical methods.

There are a number of reasons why the argument over tonsillectomy as a predisposing cause of poliomyelitis has remained so confused. One of the most important, if also the least tangible, is the emotionalism apparently inherent in any discussion of this disease. Part of the responsibility for this situation must be attributed to the fund-raising activities of the National Foundation for Infantile Paralysis, valuable as the work of this organization has been. The publicity which attended the recent introduction of the Salk vaccine and the difficulties which promptly arose over its administration have done nothing to clarify the confusion. The single death during the testing period followed a tonsillectomy and occurred two days after the child had received the second injection of vaccine. The fact that poliomyelitis was already prevalent in the area, and the further fact that the child probably already had the disease when his tonsils were removed, have been lost sight of. The circumstances, as many otolaryngologists would testify, have brought some parents close to hysteria.

The chief reason, of course, why the possible tonsillectomy-poliomyelitis sequence has not been clarified is that we are still completely ignorant of many important facts about this disease. We know that the subclinical disease is far more frequent than clinical poliomyelitis, though the suggested ratios range from 1:100 to 1:600. We know that poliomyelitis is caused by a virus which is a total parasite and which has a specific affinity for neural tissue. We still know nothing really definitive about the mode of transmission. Many other immunologic factors are still obscure. We are not even certain that all the strains of the causative virus have been discovered. The developments of the last few years give hope that the answers to these and other questions will not be too long delayed. Until they are at hand, however, we can scarcely expect the disputed relationship between tonsillectomy and subsequent poliomyelitis to be completely clarified.

Meantime, the otolaryngologist will continue to be confronted with the necessity of performing tonsillectomy, particularly in young children, when the operation is indicated. No matter on what side of the argument he stands, he must bear in mind the possible subsequent development of poliomyelitis and his possible responsibility for its occurrence, whether the disease was pre-existent, or fortuitous, or actually the result of the operation.

II. PURPOSE OF THE PRESENTATION.

An inclusive review of the literature of poliomyelitis would be an obviously impossible task. It has grown to monumental proportions and most of it either has nothing to do with tonsillectomy or mentions the operation only incidentally. The literature of post-tonsillectomy poliomyelitis is also large, but a selective review permits a record and evaluation of contributions that may throw some light upon this problem.

In addition to such a selective review, this presentation has another purpose; to put on record the tonsillectomy-poliomyelitis statistics for the period 1949-1953 in a community in the Southwestern part of the United States. The justification for the analysis is that only by the accumulation of data in adequate numbers from all parts of the country for periods of several years can this problem be resolved within the limits of our present knowledge.

It seems fair to say that the numerous general articles on post-tonsillectomy poliomyelitis represent no real contribution to the subject. For the most part, they amount to little more than expressions of the writers' opinions, secured from the data of other observers, or sometimes, from the unsubstantiated opinions of other writers. These articles contain no statistical evidence to support the points of view advanced, and for the most part, there is little evidence of the authors' clinical experience. They have been carefully reviewed in the preparation of this thesis, but for the most part they are neither recorded in the list of references nor commented upon further. The reviews of the subject which have appeared at intervals in the literature, such as Singleton's[2,3] excellent analyses, are also omitted, since any material of value in them is utilized in the course of the presentation.

III. EXPERIMENTAL STUDIES.

It is now generally agreed that experimental poliomyelitis in the monkey differs sufficiently from the natural disease in the human subject to make it necessary to be cautious in drawing firm conclusions from experimental studies;[4] nonetheless, certain experimental observations which are pertinent to the subject of post-tonsillectomy poliomyelitis should be briefly mentioned:

Nasopharyngeal Transmission of Poliomyelitis.

In 1910, Flexner and Lewis[5] inoculated the brains of monkeys with preparations of the nasopharyngeal mucosa of infected monkeys. No stool studies were made. The injected monkeys promptly developed the typical paralytic type of poliomyelitis, thus proving that the mucous membrane of the nasopharynx of the infected animals contained the virus of the disease. These animals were sacrificed, and a second set of monkeys was injected with preparations of their spinal fluids and brains. The virus was recovered in the nasopharyngeal mucosa of the injected animals. These observations permitted the conclusion that the virus of poliomyelitis may travel from the nasal mucosa to the brain and spinal fluid, or in the reverse direction.

In 1911, Landsteiner, Levaditi and Danulesco[6] were able to produce poliomyelitis by submucosal injection of the virus into the tonsillar area. The following year Levaditi and Danulesco[7] failed to produce the disease merely by rubbing or painting the tonsillar area with the virus, though application of the same preparation to the nasal mucosa produced typical disease. As Aycock[4] observed, neither set of experiments proves very much, since "monkeys develop poliomyelitis with greater or less frequency resulting from numerous routes of inoculation."

In 1912, Flexner and Clark[3] swabbed the nasal mucosa of a healthy monkey with a preparation of ground spinal cord secured from a recently paralyzed monkey. The treated animal was sacrificed at the end of 48 hours and suspensions of the olfactory lobes, the medulla, and various segments of the cord were injected into the brain and peritoneal cavity of three other monkeys. The animal injected with a suspension of the olfactory lobes died with paralytic poliomyelitis on the twelfth day. The other animals remained well. This experiment apparently showed that the virus of poliomyelitis ascends by a direct lymphatic path from the nasal mucosa to the olfactory lobe before it reaches the medulla and cord, and that infection does not occur by way of the blood stream.

In 1938, Sabin[9] reported a series of experiments on monkeys which may be briefly summarized as follows:

1. When preparations of the virus were applied to wounds created in the oropharynx by tonsillectomy, the monkeys remained well, whether the application was by swabbing, spraying, or nasal instillation.

2. When the virus was injected subcutaneously, intradermally or superficially into the post-tonsillectomy fossa, animals injected with preparations of low potency remained well. When larger quantities of a more potent preparation were injected, about a third developed the disease.

3. In this experiment, an attempt was made to re-create the conditions presumably present when the throat harbors the virus of poliomyelitis before tonsillectomy. Tonsillectomy was performed immediately after the tonsillar area had been injected with a preparation of the virus, in an endeavor to determine whether exposure of the cut nerve endings or cutting through tissue which contained the virus would stimulate the development of the disease. Only the animals in which the injection had been made deep to the bone developed poliomyelitis. The others remained well.

4. When the tonsillar area was injected before operation, as in the preceding experiment, and the fossa was also swabbed with virus immediately after the tonsillectomy, about a third of the animals succumbed to the disease.

These experiments showed several things:

1. In the monkey, the animal which most nearly resembles the human subject, the olfactory mucosa is the only known peripheral tissue which provides a portal of entry to the central nervous system by mere transitory contact with the virus of poliomyelitis.

2. Mere transitory contact between the virus and the normal or injured pharynx or tonsils is not sufficient to produce the disease.

3. Poliomyelitis apparently develops after tonsillectomy when the virus is already present at operation, rather than as the result of contamination after operation. These observations support the concept that bulbar poliomyelitis which follows tonsillectomy within a period corresponding to the incubation period of the disease is preexistent. They do not support the concept that the disease is the result of postoperative infection.

Sabin's[10] later observations on olfactory bulbs from patients with poliomyelitis, including studies of serial sections, failed to reveal the specific lesions which one might expect to find if the route of infection had been via the olfactory pathway. Previous investigators had considered the nasopharyngeal route the most probable natural portal of entry; more recent observers had considered only the olfactory mucosa of the nasopharynx as the route of infection. In Sabin's opinion, the tonsillopharyngeal route could not be regarded as the natural route. In fact, as he said, no direct data were available to show how the virus of poliomyelitis enters the central nervous system from the olfactory area or from anywhere else in the human body.

In 1939, using macacus cynomolgus monkeys, Burnet, Jackson and Robertson[11] produced the disease in seven of 19 animals by swabbing the tonsillopharyngeal region with poliomyelitis virus.

In 1942, Toomey and Krill,[12] after performing tonsillectomy on six macacus rhesus monkeys, kept the area between the cupped pillars flooded for five days with a 10 per cent suspension of poliomyelitis virus. As in Sabin's similar studies, the animals remained well, apparently because the contamination was entirely postoperative, was only superficial, and was accomplished with preparations of low potency.

In 1951, Faber, McNaught, Silverberg and Dong[13] conducted a series of experiments on cynomolgus monkeys. All the animals were prepared by division of the olfactory tracts, to preclude entry of the infection through the olfactory nerves. The results were as follows:

1. The tonsils were enucleated immediately after application of the virus to the pharynx. Paralysis ensued in four animals within seven to 10 days; in one instance it was limited to the bulbar and in three to the cervical innervations.

2. Tonsillectomy was performed three days after the virus had been injected into the thalamus. In this group of animals paralysis ensued five days after inoculation and two days or slightly less after tonsillectomy. The entire cord as well as the bulbar center was involved. The rapidity with which poliomyelitis developed in this experiment suggested to Faber and his associates that the physical stress of operation greatly enhanced the speed and severity, and to some extent the localization, of the infectious process. They believed that the results could be attributed to the introduction of the infection into the divided nerves before the endings could be covered or sealed off by blood. In an earlier publication, this group had not regarded the presence of "bare" nerve endings as essential to penetration of the virus.[11]

3. In this experiment the virus was applied to the pharynx, as in the first experiment; but, before tonsillectomy was performed, the area was swabbed with a 2 per cent solution of tincture of iodine. The iodine application was made 30 seconds after the application of the virus and 60 seconds before tonsillectomy. All six animals remained well. This method does not seem to have been tested in human subjects, nor is there any record of laboratory studies to determine whether tincture of iodine in this concentration will destroy the virus of poliomyelitis.

4. Control animals were treated simply by swabbing the surfaces of the mouth and pharynx with the virus; tonsillectomy was not performed. Five of the seven developed poliomyelitis, in one instance of the bulbar type.

In 1946, Faber and Silverberg[15] had made a study of eight autopsied cases of poliomyelitis to determine the possible portals of entry of the virus. Their theory was that the primary invasion occurs through nerve fibers with peripheral endings on mucous surfaces, the virus being conveyed in a central direction to the regional ganglia and thence to the central nervous system. Although they believed that entry could be effected via the mucosa of the nose, pharynx, trachea, esophagus, intestine and bronchus, they regarded the pharynx as a particularly favorable site, though their reasons for this point of view are not particularly convincing.

Neutralizing Properties of the Nasopharyngeal Mucosa.

In 1917, Amoss and Taylor,[16] in a series of 56 experiments, showed that washings of the nasal and pharyngeal mucosa apparently possess definite power to inactivate or neutralize the active virus of poliomyelitis. The ability varied from person to person, and inflammatory conditions of the upper air passages tended to destroy or diminish it. In the opinion of these observers, their results explained why the olfactory bulb and nerve, along with their centers, are so seldom involved at autopsy in poliomyelitis.

In 1935, Lennette and Hudson,[17] after sectioning the olfactory tract of five monkeys, sprayed the nasal cavity with a preparation of virus. All the animals remained well. When the same experiment was carried out on nine intact monkeys, all died of poliomyelitis. The five sectioned monkeys also remained well after intravenous injections of 10 cc. of 10 per cent preparations of the virus. When the same experiment was carried out on five intact monkeys, four died. Lennette and Hudson concluded from these studies that virus is excreted from the blood stream onto the nasal mucosa, whence it enters the endings of the olfactory nerve and then migrates to the central nervous system.

In 1939, experimental studies by Burnet, Lush and Jackson[18] demonstrated only slight "virucidal" activity against the virus of poliomyelitis in the nasal secretions. The finding, however, was thought to be significant, and to offer a possible explanation for the infrequent changes found at autopsy in the olfactory nerve and its centers.

In 1941, Allen and Clark,[19] working along the same line, studied the ability of soluble components of the olfactory mucosa to neutralize the virus of poliomyelitis. Consistently positive results were not obtained.

IV. STATISTICAL STUDIES.

In 1942, after a review of the literature and a personal survey of his own, which will be commented on later, Seydell[20] remarked that it was impossible to draw any definite conclusions relative to the relationship of tonsillectomy and poliomyelitis from his own survey or from the literature. Much the same situation, as has already been intimated, still prevails.

On the surface, it might seem that nothing would be simpler than to construct a table which would show, for each report in the literature, such essential data as *1.* the number of cases of poliomyelitis in the series; *2.* the number of tonsillectomies performed over the same period; *3.* the period covered by the study; *4.* the number of cases of poliomyelitis which followed tonsillectomy; *5.* the number of cases of the bulbar variety of disease in the total series; and 6. the number of post-tonsillectomy bulbar cases. The construction of a table that is complete in all these data is an impossible task. The table which attempts to present them (see Table I) has been permitted to stand, because it conserves space and avoids repetition. Actually, aside from the blanks which indicate data not stated, or so stated that they are not useful for tabular purposes, the presentation is misleading in a number of respects:

1. Only a very few of the reports contained the essential data just outlined. Reports that contained them all were exceptional.

2. In many instances complete data were lacking as to the tonsil status of a considerable number of patients, and sometimes of most patients. This information would seem essential to any discussion of the subject.

3. In only six of the 19 recorded series (see Table II) was there any attempt to supply the number of tonsillectomies performed over the period in which the cases of poliomyelitis occurred.

4. In many instances the studies were limited to special age

TABLE I

Poliomyelitis After Tonsillectomy Essential Data From Selected Reported Cases

Date, Authors	Period Surveyed	Location	Poliomyelitis Cases	Poliomyelitis Bulbar	Tonsillectomy No	Tonsillectomy Bulbar	Tonsillectomy Yes	Bulbar	Remarks
1929, Aycock and Luther[1]	1927 28	Vermont, Mass	714	--	497		217	--	
1937, Fisher and Stillerman[22]	1935	New York City	686	55			10	5	Within 1 month
1938, Eley and Flake[23]	--	Boston	418	131	224	40	194	91	17 in 1 month
1938 Fischer, et al[24]	1937	Toronto	507	63	267	20	231	43	8 tonsillectomies 1937, 4 bulbar Age 3 12
1941 Top, Vaughan[25]	1939	Detroit	521		306	4 2	215	27%	
1941, Helms[26]	1937 38	New South Wales	711	91			7	3	Within 7 22 days
1942, Seydell[20]	1940	Kansas	537		307	53	99	46	Within 1 month
1944, Lucchesi and LaBoccetta[27]	1937 42	Philadelphia	432	76	263	25	164	51	Including bulbo spinal type
1944, Howard[28]	1937 43	Cincinnati	257		--			6	
1945, Anderson J A[29]	1943	Utah	100				17	17	Within 1 month Only summer months surveyed

TABLE I — Continued.

Date, Authors	Period Surveyed	Location	Poliomyelitis		Tonsillectomy				Remarks
			Cases	Bulbar	No.	Bulbar	Yes	Bulbar	
1946, Roberts[30]	1946	Connecticut	180	......	49	......	56	5	Within 1 month. Only summer months surveyed.
1947, Pedersen[31]	1941-45	San Francisco	492	......	206	11	148	24	11 in 2 months
1947, Winborn and Stansbury[32]	1946	Dallas	134	......	44%	5	66%	17	January-August surveyed.
1949, Cunning[33]	1946	13 States	2,290	382	......	......	56	18	1-2 months.
	1947	23 States	4,331	509	......	......	24	6	
	1948	36 States	10,624	1.481	......	......	38	27	
1950, Anderson, G. W., et al.[34]	1946	Minnesota	2,709	......	......	......	37	16	1-3 months.
1952, Miller[35]	1951	Los Angeles	675	......	469	61	206	53	
1952, Miller[36]	1949	Los Angeles	1,229	......	......	......	5	2	Within 1 month.
	1950	Los Angeles	984	......	......	......	8	......	
	1951	Los Angeles	1,388	......	......	......	7	......	
1952, Top[37]	1940-49	Detroit	1,947	......	936	......	1,011	......	

* A number of reports were necessarily omitted from this table because the data were so stated that they were not useful for these purposes. No attempt has been made to derive data in any category by combinations of statistics or otherwise. Since the data stated were not always entirely clear, there is no doubt this table contains some errors.

groups, or to certain months of the year, the ages and months varying from report to report. The individual author has, of course, the right to set up the framework of his own study, but individual vagaries make cumulative studies practically impossible.

5. Some series include all patients who developed poliomyelitis after removal of their tonsils, regardless of the time interval. Other series give the information only for tonsillectomies performed one or two months before the poliomyelitis developed.

6. As has been suggested, the diagnosis is probably questionable in at least some of the cases of poliomyelitis, as well as the post-tonsillectomy cases. Only the report by Siegel and his group[21] specifies methods of confirmation of the diagnosis in the post-tonsillectomy cases.

7. In a sense, much of the material studied is weighted for the reason that it is derived entirely from hospital cases. As a number of observers have pointed out, hospital facilities are overcrowded during epidemic years, and only patients with the most severe types of disease are admitted. This fact undoubtedly explains the abnormally high incidence of bulbar poliomyelitis in certain series.

8. The data in the tonsillectomy table (see Table II) are more accurate and certainly constitute a fairer presentation of the tonsillectomy-poliomyelitis sequence than the data in Table I. Table II, however, also suffers from certain defects. The first is that some of the material was collected by questionnaire. The returns were surprisingly good, but since the diagnosis, in effect, rests upon the patient's accuracy, this is still not the best way to secure an overall picture. The second defect is that tonsillectomies done in private offices are not included in recorded series. At first glance, this might seem a trivial omission. Frequently it is not. It was surprising to find that 23 of the post-tonsillectomy cases collected by Siegel and his group[21,41] in New York City followed operations done in private offices. These cases are necessarily omitted in detailed analyses. Finally, because the data produced were obviously incomplete, some of the tonsillectomy series are esti-

TABLE II.

Poliomyetis After Tonsillectomy. Collected Series of Tonsillectomies.

Date	Author	Period Surveyed	Location	Tonsillectomy	Poliomyelitis Cases	Poliomyelitis Bulbar
1944	Page[38]	1937-39-41	New York City	8,915	1	0
1945	Anderson, J. A.[29]	1943	Utah	4,199	17	17
1946	Roberts[30]	1943	Connecticut	6,610	......	5
1946	Cunning[39]	1943-45	New York City	2,289	3	0
1947	Pedersen[31]	1941-45	San Francisco	57,796	......	......
1948	Kinney[40]	1946-47	Ohio	9,873	237	......
				12,413	184	1
1950	Anderson, G. W., et al.[34]	1946	Minnesota	7,620	37	......
1951	Siegel, et al.[21]	1944-49	New York City	53,007	30	17
1952	Miller[36]	1949	Los Angeles	14,812	10	......
		1950	Los Angeles	14,586	8	......
		1951	Los Angeles	14,544	10	......

mates, made by competent statisticians, for the most part, but nonetheless estimates.

9. None of these data makes allowances, and for practical reasons cannot, for variations in the virulence of the poliomyelitis virus from year to year and from locality to locality. They also make no allowances for host resistance.

What all of this adds up to, to put it quite bluntly, is that as matters now stand, we have very little reliable information concerning the possible relationship of tonsillectomy and poliomyelitis. The comment, in fact, seems perfectly fair that many reports amount to little more than "speculative playing with numbers."

V. COMMENTS ON RECORDED SERIES.

Sheppard,[42] in 1910, was the first to call attention to the occurrence of poliomyelitis after tonsillectomy. Thirty-one years later, in 1941, the simultaneous occurrence of five cases in siblings submitted to tonsillectomy on the same day[43,44] focussed the attention of both profession and public on the possible relationship of operation and disease.

Sheppard's report was an epidemiologic study of 200 cases of poliomyelitis in Springfield, Mass. In two cases tonsillectomy had preceded the illness. In one case the operation had apparently been performed at the onset of the infection; in the other, bulbar poliomyelitis followed tonsillectomy within the accepted incubation period.

Aycock[4] considers it interesting that Sheppard "intuitively" singled out tonsillectomy as a possible etiologic factor in poliomyelitis. His (Sheppard's) reasoning was that tonsil and adenoid tissue might offer resistance to microbic invasion. If so, he said, "it might seem on *a priori* grounds injudicious to remove an inflamed tonsil, especially in the presence of an acute epidemic infection characterized in many instances by involvement of the upper respiratory passage." Be this as it may, two instances of poliomyelitis in 700 tonsillectomies does not seem highly significant.

In the so-called "K" episode, as already mentioned, five children living in Akron contracted poliomyelitis after tonsillectomies performed on the same day, and three of the five died. The sixth and youngest child, who was not operated on, did not contract the disease, and the parents also remained well.

Although the details of the epidemiologic study by Francis and his associates[14] are too numerous to be repeated here; the important fact is that all of these children had contacts with young relatives who had positive stools, and that there was also contact with a relative who later apparently had a mild case of poliomyelitis. In the preliminary report of this episode, immediately after it occurred, Krill and Toomey[13] drew no conclusions as to the cause of the illnesses. Francis and his associates,[14] in their epidemiologic study the following year, stated that the operations had precipitated the attacks.

One of the most remarkable features of this episode is that Akron in 1941 had been singularly free from poliomyelitis. There had been only two cases in the city preceding the experience of the "K" family, and the "K" children had had no contact with these patients. Only six other cases occurred after the "K" episode. The four oldest children underwent teeth extractions in addition to adenotonsillectomy; in the reports of the experience no special attention is paid to the additional stress and strain which these procedures undoubtedly caused. It is particularly regrettable that background information on the number of tonsillectomies performed in Akron in the summer of 1941 was not supplied in any of the reports. Incidentally, as this thesis is being completed, newspapers and radios are carrying reports of a family in the Middle West in which eight of the 10 children are ill with poliomyelitis; antecedent tonsillectomy is not mentioned in any of the news items.

The essential data in Tables I and II are self-evident and need no special comment. It should be pointed out, however, that the percentage of tonsillectomized cases varies from series to series, depending upon several considerations:

1. The age of the patient included in the series. This is particularly true of the earlier reports, which were made when tonsillectomy was a far more frequent operation than it is now.

2. The period of the year included in the survey. In some series, the data are limited to the epidemic months. This makes a difference in the later reports, since in a number of hospitals, by agreement or direction, elective surgery was almost entirely discontinued at these times.

3. The number of cases of poliomyelitis which had to be excluded from the analysis because the tonsil status was not known. In some instances this necessity reduced the original statistics by a third or more.

The reported incidence for tonsillectomy varies rather widely. Miller[36] found it to be 1:135 of the Los Angeles population in the two-to-nine-year-old group. Kinney[10] found it 1:113 in a county in Ohio. Lederer and Grossman[45] mention a ratio of 1:75 for the total population of the county. Roberts[30] uses a figure of 2,000,000 tonsillectomies annually in the United States. Top and Vaughan[25] remark that the incidence of the operation varies with the social and economic status of the population surveyed.

Miller's[36] cumulative survey of poliomyelitis after tonsillectomy for the years 1949, 1950 and 1951 in Los Angeles and Los Angeles County illustrates a dispassionate, scientific approach to the whole problem. For this three-year period he reported 3,601 cases of poliomyelitis in an area population of about 4,000,000 persons. Twenty followed tonsillectomy (see Table I).

In 1949 the ratio of poliomyelitis to the two-to-eight-year-old population was 1:1,352. In 1950 the ratio was 1:1,275, and in 1951 it was 1:999. In 1949, the estimated number of tonsil operations in this age group was 14,812; the expected cases of poliomyelitis after tonsillectomy 10.9 and the actual number 10. In 1950, the figures were, respectively, 14,586, 11.5 and eight. In 1951 the figures were, respectively, 14,544, 11.5 and 10. In short, the actual and expected incidences are practically the same. Curiously, there were fewer post-tonsillectomy cases in the months when poliomyelitis was most frequent than there were in the remainder of the year.

Miller[36] also studied the tonsillectomy status of 675 patients with poliomyelitis in Los Angeles County (see Table II). His figures for tonsillectomy, 30 per cent, are in general agreement with those of other writers, who set the incidence of tonsillectomy in youth at about a third. The rate of poliomyelitis after tonsillectomy thus seems to follow the rate of tonsillectomy in the general population. Bulbar poliomyelitis, however, appeared more often and was likely to be more severe in recently tonsillectomized patients.

Tonsillectomy Studies.

The first serious attempt to determine the incidence of poliomyelitis after tonsillectomy, instead of merely stating the number of tonsillectomized patients in series of cases of poliomyelitis, was made by Page,[38] in 1944, from the Manhattan Eye, Ear and Throat Hospital (see Table II). In 8,915 replies (out of 27,849 patients circularized, which is a very good response), he found that there had been only one instance of "infantile paralysis" within a month after tonsillectomy. Page's investigation covered the years 1937, 1939 and 1941, in all of which poliomyelitis was prevalent.

In 1951 Siegel and his associates[11] carried out a similar investigation in the same hospital for the period 1944-1949. In the 6,524 replies which they received to their questionnaire they found that 30 cases of poliomyelitis, 17 of the bulbar type, had occurred within a month after operation.

Roberts,[30] in 1946, arrived at the relative incidence of post-tonsillectomy poliomyelitis by a series of calculations. If, he said, government records could be accepted, that 2,000,000 tonsillectomies are done annually, then 70,000,000 would have been done between 1910, when the relationship was first observed, and 1945. A review of the literature up to 1943 revealed 274 cases of post-tonsillectomy poliomyelitis, a ratio of one to each 255,474 operations. Since no case of post-tonsillectomy poliomyelitis had been reported over 18 years of age, the tonsillectomy figure should be reduced to 52,500,000, which would be a ratio of 1:191,605. On the assumption that 53 per cent of all tonsillectomies are done in the July-October period, the figures are further reduced to 27,825,000 and the ratio to 1:101,551. Since the average incidence of poliomyelitis in the general population, according to figures supplied by the Bridgeport Board of Health, is 1:3.250, the incidence in the general population is still [illegible]1 times more frequent than after recent tonsillectomy, even assuming that all 274 cases reported after tonsillectomy occurred in the so-called poliomyelitis months (which they did not). It is doubtful that a statistician would be willing to accept this reasoning, but the calculations are cited for what they are worth, as an illustration of one kind of attempt that has been made to reach a conclusion in this matter.

VI. CONCLUSIONS FROM STATISTICAL SURVEY.

Conclusions drawn from these statistical surveys are, naturally, widely divergent. Some of them seem justified by the studies on which they are based; others do not. Certain of them may be mentioned.

The earliest series of cases reported from the special viewpoint of a poliomyelitis-tonsillectomy relationship was by Aycock and Luther,[1] in 1928 (see Table I). From this investigation, they concluded that it could not be stated with certainty that the preceding tonsillectomy was responsible for the localization of the paralysis. In 1942, however, after a survey of the literature and a study of additional data, Aycock[4] concluded that a causal relationship does exist between the removal of the tonsils and the occurrence of poliomyelitis within a specific interval. He also noted that this concept was in line with experimental observations, which showed that bulbar poliomyelitis was likely to occur after tonsillectomy when the virus was already present at the time of operation. His data also showed that bulbar poliomyelitis was more frequent at all ages when the tonsils had been removed. He was not willing to conclude that the absence of the tonsils predisposed to clinical poliomyelitis, even though tonsillectomy had been performed more frequently in patients with poliomyelitis than in controls studied in the same area. Aycock felt that because of numerical considerations (the fact that roughly 30 per cent of the population undergoes tonsillectomy at some time, whereas poliomyelitis occurs in only a fraction of 1 per cent), it would be difficult to say to what extent the operation should be avoided because of the relatively small risk of developing bulbar poliomyelitis. The risk should be recognized, but the decision for or against operation should be upon the basis of the indications in the individual case.

Toomey and Krill,[46] who reported a combined series of 430 cases, from Akron and Cleveland, concluded that more patients contracted bulbar poliomyelitis after tonsillectomy than could be explained by mere chance or random sampling. From the standpoint of the relationship of the mortality to the tonsil status, it seemed to make little difference whether the tonsils were in or out; in fact, if mortality were the only criterion, it would seem that death was less apt to occur in previously tonsillectomized patients.

Siegel and his associates,[21,11] by statistical methods, determined that the risk of contracting poliomyelitis was significantly greater than the expected risk within a month after operation, but observed no increase in the incidence of bulbar paralysis after six months had passed. On the other hand, differences in the numbers of observed and expected cases were often not great enough to rule out sampling variations, while in other instances the figures were at the borderline of statistical significance. This group considered children up to nine years of age more vulnerable to poliomyelitis when they were tonsillectomized in the spring, especially in June, and regarded January, February and March as the safest period of electiv surgery in New York.

Langworthy,[17] while he would naturally be unwilling to operate in the midst of an epidemic of poliomyelitis, who provided no independent statistical data, took rather violent exception to the assumption of a tonsillectomy-poliomyelitis relationship. He saw no justification for postponing tonsillectomy when it was needed because a few cases of poliomyelitis existed in the community. He mentioned the harm that delay might do to children with bad tonsils, malnutrition, repeated aural and upper respiratory infections, and infections of the cervical glands.

Wesselhoeft,[48] who also provided no supporting data, had originally refused to perform tonsillectomy during the Summer months. By 1948 he had changed his mind and felt that in the New England states the ideal time for the operation was immediately after the close of school. He would continue to operate through the Summer if no cases were reported from the community and there was no known possibility of exposure from the outside. He justified his stand, in part, by other dangers, such as upper respiratory infections, when the operation was performed during the Winter.

Miller,[36] whose sound statistical surveys have been quoted at considerable length, concluded that during the years of his study the actual number of cases and the expected number were so close to each other that the differences were of no significance and tonsillectomy could be assumed to have played no part in the development of the disease during the years in question. In his survey of the 1949 material Miller mentioned that twice as many children had undergone tooth extraction as tonsillectomy. He felt that there was nothing in the figures to indicate that tonsillectomy need be discontinued unless one happened to believe that its performance any number of months before the onset of the infection could be a factor in its development.

Cunning,[33,39] whose yearly surveys in 1947, 1948 and 1949 were conducted with great care, and were as nearly nationwide as he could make them, did not feel that his collected data justified a statement of a causal relationship between poliomyelitis and tonsillectomy. He realized that he was in disagreement with many of his associates when he took this position. In his own opinion, acceptance of a predisposition to the disaese after tonsillectomy was based in part on experimental evidence and in part on limited clinical evidence and surveys. He did not think that tonsillectomy should be postponed indefinitely merely because poliomyelitis is prevalent in the Summer months, though as a matter of common sense, elective surgery should be discontinued promptly if there is a rise in the incidence. Unless conclusive evidence is forthcoming as to the relationship, the otolaryngologist should decide whether hardship to the patient by delay and postponment is greater than the still undetermined risk of increasing his susceptibility to poliomyelitis by performing a tonsillectomy. This is a reasonable and sound position, based on a wide clinical experience and supported by ample statistical data, personally collected and surveyed.

VII. CONCEPTS OF THE TONSILLECTOMY-POLIOMYELITIS RELATIONSHIP.

In 1917, on the basis of his own experience and research, Seydell[49] took the position that the tonsils were a harbor of infection in poliomyelitis. Like Rosenow, he believed that tonsillectomy in the course of the disease was helpful. In this same communication Seydell cited the studies of Vaughan, Vaughan and Palmer:[50] During the 1916 epidemic in New York, a large number of the children affected had hypertrophied tonsils, or adenoids, or both. The disease was uncommon when the tonsils had been removed. After the epidemic, in which 8,928 cases occurred, an investigation in the public schools showed that not a single case had occurred in 1,404 tonsillectomized children, though in 18 instances there has been cases in the same family, and in 93 cases in the same house. The concept of a possible protective effect of tonsillectomy has, of course, long since been discarded.

Since the virus of poliomyelitis travels along the nerves, and since it may have an obligate affinity for the gray fibers of either medullated or unmedullated nerves, it may be that when the axis is exposed during tonsillectomy, the virus is more easily absorbed.[46] In the healing process, new vascular networks are laid down, accompanied by the deposition of new unmedullated fibers, which readily absorb the virus. On the other hand, most pathologists believe that by the end of 24 hours after tonsillectomy, a local tissue reaction has developed which would prevent the invasion of most pathogenic organisms.

To Seydell[20] it seems more unlikely that the virus can penetrate the scar tissue which develops in the tonsillar fossa after tonsillectomy than that it can invade the loose lymphatic tissue of the tonsil and its lymph and blood vessels.

Aycock[4] discussed the possible relationship of poliomyelitis and tonsillectomy from the standpoint of what he calls autarcesis, that is, resistance to the host, independent of immunity from exposure to the virus. He believes that the selectivity of the development of bulbar poliomyelitis after tonsillectomy is best explained in this manner, on the assumption that some added circumstance plays a part in determining whether clinical or subclinical disease will follow exposure to the virus. This selectivity, and the relatively more frequent occurrence of bulbar disease in tonsillectomized persons, in his opinion, comprise a demonstration that the nasopharyngeal mucosa may be the *locus* of at least one added circumstance that determines the outcome of exposure. His conclusion is that since tonsillectomy is practically always elective as to time, changing the season when it is done so that it will not coincide with the season of poliomyelitis prevalence, could eliminate many cases of a highly fatal form of the disease.

McCormick[51] postulated a possible relationship between the basic hypertrophy of tonsils, adenoids and other lymphatic structures often present before tonsillectomy and the rather general lymphatic hypertrophy frequently present in poliomyelitis. He linked up this observation with the lymphatic hypertrophy frequently found in nutritional deficiency, especially vitamin-*B* deficiency. In the 1937 epidemic in Toronto it was found that among persons on relief, who had a low vitamin-*B* intake, there were four times as many cases of poliomyelitis as in the rest of the population.

McCormick also mentions other possible factors, aside from a direct tonsillectomy-poliomyelitis relationship, including the shock of the operative procedure, and the anoxia produced by anesthesia and the barbiturates often used in preoperative preparation. He marshals considerable evidence for this theory, including the fact that brain tissue deficient in vitamin-*B* absorbs oxygen more slowly than normal brain tissue, and the further fact, demonstrated by Van Liere, that severe cerebral anoxia can produce an ascending type of paralysis, with eventually, bulbar and cerebral involvement. McCormick's conclusion is that neuromuscular anoxia, associated with either a deficient nutritional status, or surgical anesthesia, or both, may supply the missing link in the etiologic relationship between tonsillectomy and poliomyelitis. In his opinion, the association may be much closer than ordinary contact.

Several observers, notably Top and Vaughan,[25,37] believe that factors other than the tonsillectomy, with its surgical wound, may play some part in the development of poliomyelitis after operation. They mention stress and strain, trauma, physical activity, the body type, endocrine changes, dietary deficiencies, pregnancy, and preceding disease. Cunning[39] argues along much the same line. He assumes that in the large number of persons who harbor the virus of poliomyelitis with few, if any, clinical manifestations, there is some unknown factor or phenomenon which maintains an equilibrium between the host and the virus, so that clinical disease does not develop. He further assumes that any disturbance which upsets this equilibrium may so accelerate the action of the virus as to provoke clinical disease. If tonsillectomy is regarded as such an activating factor, then one must also take into account the attendant surgical shock and the effects of anesthesia, both of which are present in all surgery, regardless of the nature or the site. Since tonsillectomy is the procedure most often performed upon children of the age group most susceptible to poliomyelitis, the emphasis has been put upon it rather than upon the surgical shock and anesthesia associated with all operations. In other words, if tonsillectomy has any influence in the development of poliomyelitis, it is probably by lowering the resistance of the body rather than by the existence of a surgical wound in a particular location.

VIII. SURVEY OF THE TONSILLECTOMY-POLIOMYELITIS SEQUENCE IN A SOUTHWESTERN COMMUNITY.

Materials and Methods.

This survey was conducted in a community in the Southwest part of the United States in which poliomyelitis has been epidemic in several recent years, but in which no previous survey has been conducted to determine the possible relationship between the disease and previous tonsillectomy.

This community, which has a population of approximately 65,000, is located in a district of 260,000 persons. Medical practice comes from a large adjacent portion of this total area, and physicians in the whole district refer many of their difficult cases to the community.

The data were derived from two sources: the record libraries of the two local hospitals which treat poliomyelitis and, when the information contained in the records was not complete, from personal letters to the patients or their parents.

These letters explained that the objective of the study was a determination of the possible relationship between tonsillectomy and poliomyelitis. They requested that the writer be informed, on an enclosed card, whether the tonsils had been removed before the developmeent of the disease, a simply worded inquiry which needed only a *yes* or *no* answer. The investigation thus avoided the pitfall inherent in inquiries which concern the development of poliomyelitis after operation. In cases which had ended fatally a carefully worded paragraph replaced the inquiry used otherwise.

The investigation covered the five-year period, 1949-1953, inclusive, and thus included both epidemic and non-epidemic years.

Essential Data.

The essential data in this investigation (see Table III) are as follows:

TABLE III

Distribution of 888 Cases of Poliomyelitis According to Tonsil Status in Community in Southwest United States 1949 1953 *

	Total Cases	Tonsils In Situ	Tonsils Removed	Status Unknown
Total Cases	888	534	254	100
Spinal	767	468	211	88
Bulbar	121	66	43	12
Deaths	11	4	4	3
Spinal	2	1		1
Bulbar	9	3	4	2

* During this same period, 2,813 tonsillectomies were performed in this community

In all, 888 cases of poliomyelitis were treated in the two hospitals in this community during the period 1949-1953. In 100 cases information as to the tonsil status could not be secured, and the analysis, therefore, chiefly concerns 788 cases, the essential facts of which are as follows:

In these 788 cases, 534 patients, 67.7 per cent, had their tonsils *in situ* when they contracted the disease.

Two hundred fifty-four, 32.3 per cent, had had their tonsils removed at some time in the past.

No patients in the series had undergone tonsillectomy within a month of their illnesses.

In 121 of the entire series of 888 cases the poliomyelitis was of the bulbar type. In 12 of the 121 cases no information could be secured as to the tonsil status of the patients.

In the remaining 109 cases of bulbar poliomyelitis, the tonsils had not been removed in 66, 60 per cent, and had been removed in 43, 40 per cent.

There were 11 deaths in the total series, four in patients whose tonsils had not been removed, four in patients who had undergone tonsillectomy, and three in patients whose status was unknown. This is an unusually low case fatality rate.

No patient in the series contracted poliomyelitis within a month of tonsillectomy. One patient, five years of age, became ill six weeks after the removal of his tonsils (1949), and another, nine years of age, six months after tonsillectomy (1950). Both patients had the spinal type of disease, and both recovered completely.

There were no instances of bulbar poliomyelitis within a six-month period after tonsillectomy.

During this same five-year period, 2,813 tonsillectomies were performed in the two hospitals surveyed. In only two instances, as just noted, did poliomyelitis develop within a six-month period after the operation, and in no instance, also as just noted, did the bulbar type of poliomyelitis occur within this postoperative period.

Yearly and Seasonal Distribution of Poliomyelitis.

The five-year seasonal distribution of the 888 cases of poliomyelitis surveyed in this study (see Fig. 1) is typical of the seasonal prevalence of the disease in this part of the United States. It is most frequent from May through September, but a scattering of cases occurs through the remainder of the year. This figure (see Fig. 1) also shows the yearly and monthly distribution of the 2,813 tonsil operations performed over the same five-year period. It will be noted that more tonsil operations are done in this community in April, May and June than at any other period of the year. The explanation of the smaller number of cases performed in July is the rather general practice, on the part of both profession and patients, of taking vacations during this month. The decrease is not due to discontinuance of tonsil operations in July. Even during the epidemic years of 1949 and 1950, particularly in 1950, throat surgery continued to be performed.

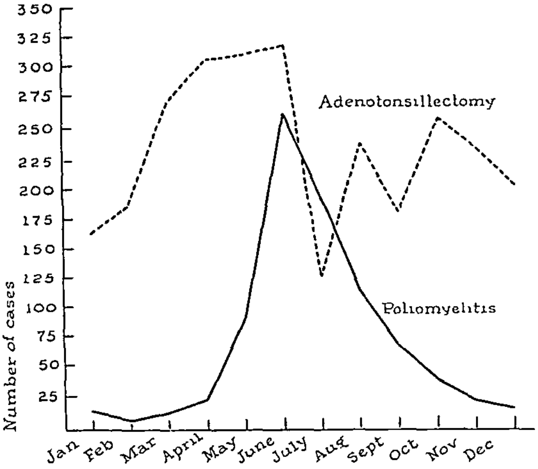

Fig 1 Seasonal distribution of 788 cases of poliomyelitis and 2,813 adenotonsillectomies in a community in the southwest United States 1949-1953.

Age Incidence.

The age incidence in these 888 cases of poliomyelitis (see Fig. 2) shows nothing unusual, and is in correspondence with observations from other parts of the country. Poliomyelitis is pre-eminently a disease of youth, and is most frequent in the five-to-10-year period, as this figure shows. Both cases of poliomyelitis after tonsillectomy occurred within this age group, but the number is too small to be worth showing graphically.

Hospital Incidence.

The distribution of all poliomyelitis cases in which the tonsil and adenoid status was known is shown separately for the two hospitals surveyed (see Table IV) chiefly as an illustration of how misleading conclusions based on small groups of figures can be. Only 92 of the 788 patients were treated in hospital "A". When this small group of cases is broken down by year and type of disease, the statistics are totally insignificant and some of the proportions are out of line. In this hospital, for instance, 38 per cent of the patients had had tonsillectomies sometime in the past. In hospital "B" the proportion was 31.4 per cent, and for the whole series it was 32.3 per cent. In hos-

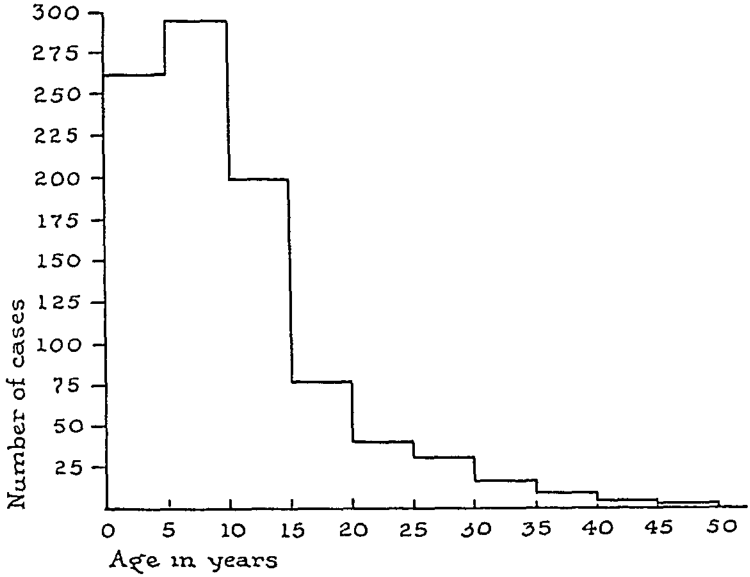

Fig. 2. Age incidence in 788 cases of poliomyelitis in a community in the southwest United States 1949-1953.

TABLE IV

Distribution by Hospital, Year, and Type of Disease in 788 Cases of Poliomyelitis with Known Tonsil Status

Tonsil Status	Hospital "A" Spinal	Hospital "A" Bulbar	Hospital "A" Total	Hospital "B" Spinal	Hospital "B" Bulbar	Hospital "B" Total	Both Hospitals Spinal	Both Hospitals Bulbar	Both Hospitals Total
1949									
In situ	25	2	27	233	35	268	258	37	295
Removed	5	6	11	90	9	99	95	15	110
1950									
In situ	6	0	6	93	12	105	99	12	111
Removed	9	1	10	45	10	55	54	11	65
1951									
In situ	5	0	5	21	10	31	26	10	36
Removed	1	0	1	14	2	16	15	2	17
1952									
In situ	13	2	15	58	2	60	71	4	75
Removed	7	3	10	33	7	40	40	10	50
1953									
In situ	3	1	4	11	2	13	14	3	17
Removed	0	3	3	7	2	9	7	5	12
Total									
In situ	52	5	57	416	61	477	468	66	534
Removed	22	13	35	189	30	219	211	43	254
Total	74	18	92	605	91	696	679	109	788

pital "A," 13 of 35 patients without tonsils contracted bulbar poliomyelitis, 37 per cent, well over twice the estimated national average of 15 per cent, but readily explained by chance in so small a group of cases. In hospital "B", 30 of 219 patients without tonsils had bulbar poliomyelitis, 13.6 per cent, and the proportion for the total series was 17 per cent, well within the general average in hospital "B", and very close to it for the whole series. Entirely erroneous conclusions might be arrived at if only the figures for hospital "A" were used. The larger figures for hospital "B" and for the combined hospitals show again that there is no relationship between tonsillectomy and poliomyelitis *per se,* nor, in this series in this part of the United States, for the period 1949-1953, any clearcut relationship between tonsillectomy and the bulbar form of the disease.

Why the incidence of bulbar disease should be higher in nonepidemic years, such as 1951 and 1953, than in epidemic years is not clear. The most obvious explanations are that only small groups of cases are involved, and that the infecting virus in those years may have been of unusual virulence.

Comment.

There is really nothing more to be said about this series of cases. It would be possible to present the figures from other various aspects, but nothing would be gained by it. The essential fact is that over a five-year period, in 788 cases of poliomyelitis in which the tonsil status was known, and in 2,813 tonsillectomies performed over the same period, only two patients contracted the disease after tonsillectomy; in neither instance earlier than six weeks postoperative, and in neither instance of the bulbar variety. The fact that tonsillectomies were performed routinely over this period, without regard to whether or not poliomyelitis was epidemic in the community, is a fact of special significance in the argument over the possible influence of previous tonsillectomy on post-tonsillectomy disease.

It is true that the case fatality rate in tonsillectomized patients with bulbar poliomyelitis, four of 43 cases, was considerably higher than the rate in patients with tonsils *in situ,* four of 66 cases. On the other hand, bulbar disease is always more serious than other varieties, and the number of cases in both tonsillectomized and non-tonsillectomized groups is too small to warrant conclusions as to risk.

IX. SUMMARY AND CONCLUSIONS.

1. A review of the literature dealing with the possible relationship of tonsillectomy and post-tonsillectomy poliomyelitis reveals two schools of thought: one group of observers believes that recently tonsillectomized patients are more susceptible to the disease, particularly the bulbar type of disease, because the virus of poliomyelitis has a readier route of entrance through the surgical wound, with its exposed nerve endings. The other school believes that tonsillectomy has no relationship to poliomyelitis which develops after it. In the opinion of this group, some cases of poliomyelitis which follow tonsillectomy are fortuitous, and others can be explained by such factors as stress, strain, surgical shock and the anoxia of general anesthesia, which are risks to be assumed in all surgery. The virulence of the causative virus is also regarded as an important consideration.

2. While the literature on the subject is extremely confused, it seems fair to say that those who believe in the causal relationship between tonsillectomy and poliomyelitis have based their opinions chiefly on experimental evidence, isolated cases, small series of cases, or series studied without due regard to statistical methods. Those who deny the relationship have investigated larger series, have studied them by sound statistical methods, and have also studied a side of the picture that was formerly ignored, namely, the incidence of poliomyelitis following large series of tonsillectomies.

3. It is unfortunate that the emotionalism inseparable from such a devastating disease as poliomyelitis has entered the picture and confused both public and profession. It has also placed a particularly heavy responsibility upon the otolaryngologist, who must decide, in the individual case, whether to perform an indicated adenotonsillectomy or to omit it for fear of the development of poliomyelitis.

4. The experimental literature is often confusing and frequently inconclusive, aside from the fact that natural and experimental poliomyelitis differ sharply from each other in some respects.

5. While a review of the clinical and statistical literature reveals widely differing viewpoints, the most valid studies indicate that the possible relationship between tonsillectomy and poliomyelitis remains to be proved.

6. An investigation of 888 cases of poliomyelitis and of 2,813 adenotonsillectomies performed over the same five-year period (1949-1953) in an area in the Southwest United States confirms the opinion that there is no causal relationship between tonsillectomy and post-tonsillectomy poliomyelitis. Only two cases of poliomyelitis occurred after tonsillectomy in the 788 patients whose tonsil status could be determined. The incidence of poliomyelitis after tonsillectomies performed during this period is thus entirely insignificant. Neither of the two cases occurred within a month of the operation, and neither was of the bulbar type. In fact, none of the 109 patients with bulbar poliomyelitis had undergone tonsillectomy within a six-month period.

7. This survey shows clearly the fallacies and pitfalls of basing conclusions upon single years and small series of cases from small institutions. When the figures in this series were broken down in this fashion, the conclusions reached were sometimes contrary to the conclusions arrived at for the total series and for the entire five-year period.

8. The soundest position in this matter was expressed by Cunning,[33] in 1949, as follows:

a. A belief that tonsillectomy is responsible for the subsequent development of poliomyelitis is based on experimental evidence and limited clinical evidence and surveys.

b. Since there is no way of foretelling when an outbreak will occur in any community, tonsillectomy should not be postponed indefinitely merely because poliomyelitis is prevalent in the summer months. Naturally, if the incidence rises, all elective surgery, including tonsillectomy, should be discontinued.

c. Until more conclusive evidence is forthcoming as to a causal relationship between tonsillectomy and post-tonsillectomy poliomyelitis, the otolaryngologist must decide, in each case that he encounters, whether the hardship to the patient *by delay and postponement would be greater than the still undetermined* risk of increasing his susceptibility to poliomyelitis by going ahead with tonsillectomy when it is indicated.

d. In short, until convincing proof is adduced that tonsillectomy increases the susceptibility to poliomyelitis, the solution *of the problem is the application of common sense to the individual* case: when poliomyelitis is prevalent in a community, elective surgery should not be performed. When it is not prevalent, *tonsillectomy may safely be performed in the cases in* which it is indicated.

9. The best way to settle this problem, in the light of present knowledge, would be to adopt Cunning's proposal that some national body undertake a controlled study, directed toward *the future and not to the past, to determine what happens after* tonsillectomy as well as how many tonsillectomized persons develop poliomyelitis. The responsibility of excessive oxygen administration for retrolental fibroplasia in premature children has just been settled by such a survey. It would be a far simpler matter to set up such an investigation of post-tonsillectomy poliomyelitis.

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Program and any further information may be obtained by communicating with Dr. Louis Joel Feit, Secretary, 66 Park Avenue, New York 16, New York.

TUMOR OF THE GANGLION NODOSUM OF THE VAGUS NERVE.*

H. J. LINN, M.D.,
and
BRUCE PROCTOR, M.D.,
Detroit, Mich.

Although tumors of the carotid body have been recognized for many years, only recently have similar tumors arising in other sites in the neck been described. The following case is reported because multiple tumors were removed from the same patient, and also because of an apparent familial relationship, and finally because of the rarity of tumors of the ganglion nodosum.

REPORT OF CASE

A white woman, single, age 25, was admitted to Detroit Memorial Hospital in June, 1951 She had noticed a small lump in the right side of her neck one year previously The tumor had increased in size during the year but had caused no symptoms

Past history was non contributory There was, however, an interesting family history A sister of the patient had a carotid body tumor removed in November, 1947, at the age of 26 years The tumor was reported to be a "malignant tumor arising in a carotid body" There was, however, no recurrence during the following three years, and review of the pathologic sections revealed the tumor to be probably benign The patient had three brothers without tumors Her mother and father were living and well One uncle had died reportedly of carcinoma of the thyroid, and one of leukemia The two uncles had six children, all without cervical tumors An aunt died of diabetes at the age of 55 years

Physical examination of the patient revealed a firm, smooth, non tender nodule, 2 cm in diameter in the right neck, which could be moved laterally, but not up and down Heart and lungs were normal, blood pressure was 112/65 mm Blood count and urinalysis were within normal limits

On June 5, 1951, under local anesthesia, a tumor of the right carotid body was removed *The blood supply seemed to come from a point 0 5 cm* above the bifurcation of the common carotid, from the medial aspect of the external carotid artery The tumor was dumb-bell shaped, measuring *22x19x17 mm, and was a typical carotid body tumor* There was no metastatic tumor in a lymph node removed from the fat, anterior

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* *From the Departments of Pathology and Oto-laryngology of Detroit* Memorial Hospital and Wayne University College of Medicine Dr Linn *is now at William Beaumont Hospital Royal Oak, Mich*

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to the carotid sheath. The postoperative course was uneventful, except for hoarseness, which slowly improved.

The patient was again admitted to Detroit Memorial Hospital on June 7, 1954, with a history of a lump in the left side of the neck for 18 months. She stated that when she was tired she "felt a strain" in the region of the lump and sometimes felt a thumping in it.

The patient had married 18 months previously, and had a child four months old. The lump in her neck had increased in size during the pregnancy.

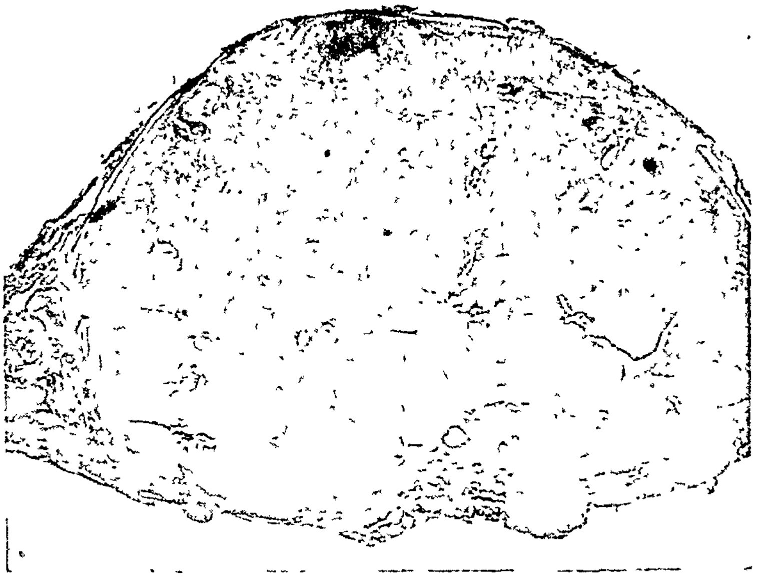

Fig. 1. Tumor of ganglion nodosum (×6).

On physical examination a mass was palpated in the left neck, anterior to the sterno-cleido-mastoid muscle, and at the angle of the jaw. As with the previous tumor, the mass could easily be moved laterally, but less freely in a vertical direction. Aside from the scar of the previously removed tumor, physical examination was negative. Urinalyses, serologic tests for syphilis and blood counts were normal.

On June 8, 1954, under endotracheal anesthesia, the tumor was removed. It was found to be a fusiform swelling of the left vagus nerve, directly posterior to the bifurcation of the carotid. The tumor was inseparable from the vagus, which was cut above and below the tumor. For a short period during the operation blood pressure readings could not be obtained.

The postoperative course was uneventful and the patient was discharged from the hospital on June 14, 1954.

PATHOLOGY REPORT.

The fusiform tumor measured 22x16x16 mm., with the vagus nerve projecting 4 mm. from each end of the tumor. The cut surface bulged slightly, and was pale pinkish-tan.

On histologic examination the tumor lay within the vagus nerve, with bundles of myelinated nerve stretched over the tumor (see Fig. 1). The latter was composed of rounded groups and cords containing four to ten cells surrounded by reticulin fibers (see Fig. 2). Numerous small capillaries with occasional larger veins coursed in the scanty connective tissue between cell groups. In several areas larger collagenous bundles were found in the tumor. The nuclei of the tumor cells were finely granular and varied considerably in size; a few nuclei were over 30 microns in diameter (see Fig. 3).

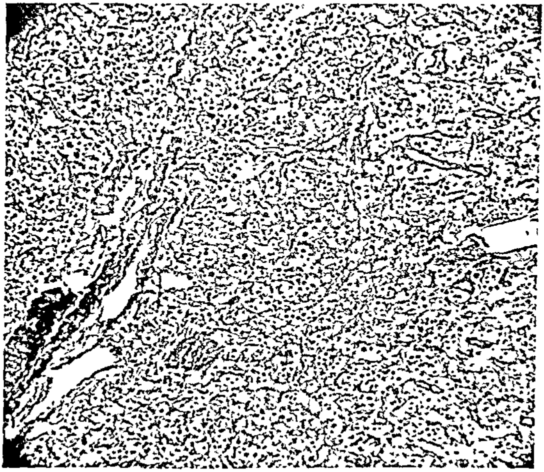

Fig. 2. Tumor of ganglion nodosum. Reticulin stain showing grouping of cells in acini and cords and intimate relationship to veins (x200).

Many cells had distinct nuceoli, but there were no mitotic figures.

The cytoplasm of the cells was often finely vacuolated. Occasional cells with pycnotic nuclei and foamy cytoplasm appeared to be degenerating, though no fragmented nuclei were found. About some of the larger venous sinuses there was a small number of lymphocytes.

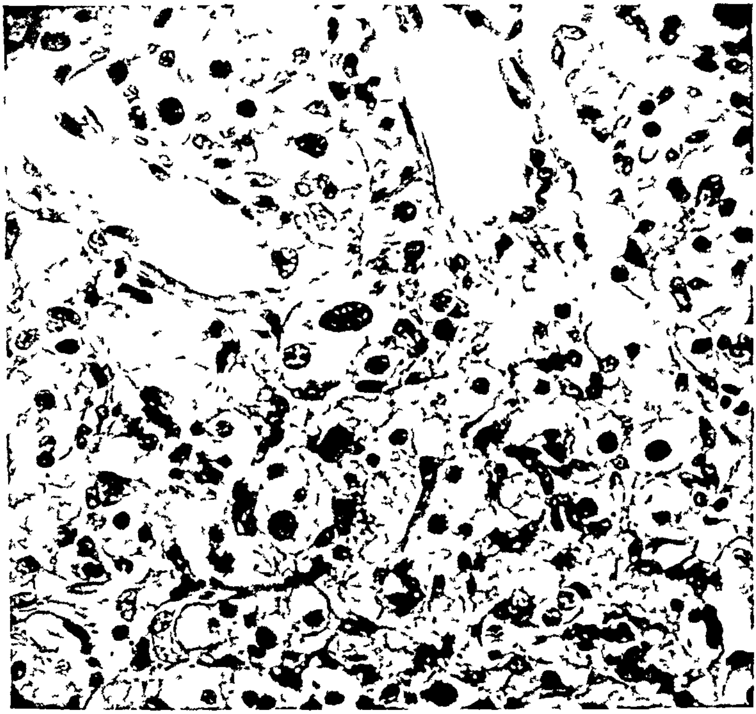

Fig 3 Tumor of ganglion nodosum There is marked variation in nuclei (Hematoxylin and Eosin ×600)

DISCUSSION.

Tumors of the carotid body and related structures, including the glomus jugulare, the paraganglion tympanicum, the ganglion nodosum of the vagus, and the aortic bodies have recently been reviewed by LeCompte[1]. These structures were once erroneously considered to be paraganglia, to be endocrine glands secreting epinephrine, and to stain green with chrome salts. They have more recently been shown to be chemoreceptor organs sensitive to changes in the pH, carbon dioxide content and oxygen tension of the blood. They are not endocrine glands, nor are they paraganglia. The histogenesis of the structures is in dispute, some claiming that they are neuroectodermal; others, that they are mesodermal in origin. The term chemodectoma has been proposed for tumors of chemoreceptor organs.

The present case is the fifth tumor reported, which arose in the ganglion nodosum of the vagus nerve[1,2]. The reported ages have been from 28 to 49 years, and all but one have been in women. In three of the five cases there were also tumors in other chemoreceptor organs. Although a familial incidence has been shown in carotid body tumors, the present case is the first in which a tumor of the ganglion nodosum occurred in a family, another member of which had a chemoreceptor tumor.

All of the five tumors were surgically excised. In one tumor it could be only incompletely surgically removed, and the patient was given X-ray therapy. He died five months later of poliomyelitis without symptoms referable to the tumor. Another patient died of bronchopneumonia and pressure of a recurrent tumor on the brain stem, 32 months after incomplete removal.

SUMMARY.

A carotid body-like tumor of the ganglion nodosum of the vagus nerve is reported, and four other similar tumors are discussed.

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A CONTRIBUTION TO THE PROBLEM OF THE BLUE EARDRUM: IDIOPATHIC HEMOTYMPANUM.

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"Blue eardrum" is a clinical symptom caused by the addition of the hue of blood to the tympanic membrane. The symptom is frequently seen in plain traumatic hemotympanum. In other cases it has been reported as a symptom of the jugular bulb protruding through a dehiscense into the middle ear, or of a hemangioma behind the eardrum; however, there remains a number of cases to which the name of idiopathic hemotympanum has been applied and which seem to represent a definite disease entity. Only 14 such cases have been reported in the literature although it is thought that many more have actually occurred. Recently Johnston[6] has given a full review of this syndrome, which is characterized by the insidious and painless onset, the copious viscid brown fluid in the middle ear, and the tendency of this fluid to reaccumulate without spontaneous cure. The minimal symptoms are in contrast to the surprising degree of conductive hearing loss. In no instance have hematologic or systemic disorders been found to explain the bleeding tendency.

Considering the few cases reported it seems justified to report another case of idiopathic hemotympanum, especially as this case offers an unusual feature, which may possibly throw some light on this rather puzzling desease.

CASE REPORT.

The patient, a white female, was seven years old when first admitted to the Roskilde County Hospital Nov. 5, 1950. Her previous history was non-contributory. Fourteen days before her admission she suffered a skull trauma by falling from a chair. There was no apparent ill effects after this event, but one week later she developed a spontaneous otorrhea from the right ear, followed within 12 hours by a complete

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facial paralysis on the same side On admission there was a copious, brownish, viscid discharge from the right ear, where the tympanic membrane was thought to be diffusely swollen, and a homolateral complete facial paralysis The mastoid region was found to be normal, and X ray examinations of the skull revealed no fracture On penicillin therapy the ear became dry in ten days, when it became apparent that the drum was dark blue, and remained essentially so despite inflations by the Politzer and the catheter method The facial paralysis improved slowly on treatments with electrical stimulation, massage, and vasodilating drugs, and upon her discharge Jan 9, 1951, it had disappeared almost completely

Three months later, after an acute upper respiratory infection, there was a recurrence of the right otorrhea, and the patient was readmitted to this department April 11, 1951, with copious, brownish, viscid discharge from the right ear, and a swollen, red tympanic membrane On penicillin therapy the ear became dry in three days On the fifth day the drum was almost normal, but on the eighth day it resumed the previous dark blue color An audiogram made at this time revealed a 20 decibel conductive hearing loss in the low frequency range for the right ear

The patient remained well until three years later, when, after a severe febrile upper respiratory infection she had earache on the right side, followed shortly by a copious brownish discharge from the ear, which was treated successfully with penicillin Fourteen days later there was recurrence of the fever, which responded to treatment with a sulfa drug, however, the purulent discharge from the nose and deafness in the right ear persisted After one month, April 8, 1954, she was readmitted to the hospital Clinical examination revealed a slightly bulging, dark blue eardrum, and some mucopurulent secretions in both nasal cavities Roentgenograms showed clouding of both maxillary sinuses, and by irrigation pus was recovered from the left side The sinusitis was treated with sulfa drugs and repeated irrigations and abated within a week As the drum remained unchanged, a generous myringotomy was performed, with removal of a large amount of brownish mucoid fluid The drainage of this fluid persisted for two days, and under treatment with repeated inflations and chloromycetin there was a definite decrease of the intense blue color of the drum Gradually, however, the fluid reaccumulated and the hearing remained severely impaired Roentgenograms of the mastoids showed a normal left side, while there was a remarkable increase of the pneumatization on the right side, with clouding of the whole cell system but without evidence of destruction

Considering the previous history it was now decided to perform a simple mastoidectomy, which was done on May 12, 1954 The mastoid was found to be most extensively pneumatized, with the cells being very large and the septa sclerotic All the cells were filled with mucoid, brownish secretions, except in the apical and deep subantral regions, where the secretions consisted mostly of pure old blood In this region also the mucous membrane was very thick, in some places resembling granulation tissue Because of the previous palsy, special attention was paid to the facial canal It was not thought advisable to open it because of the risk of a recurrence of the palsy, but the osseous covering was friable and in some places not very well marked Bleeding was only slight, and no area was encountered resembling angioma, nor were there any signs of a previous fracture On termination of the operation all cells and mucous membrane had been removed and there remained a very large cavity The postauricular incision was closed after insertion of a rubber tube into the antrum

Histological examination of a tissue specimen from the subantral region showed small tissue fragments, consisting essentially of densely cellular, proliferating tissue with very few fibrils, and only moderately vascularized. It contained numerous cholesterol crystals, some blood pigment, and a considerable number of foreign body giant cells. The intermediate osseous tissue appeared essentially normal. There was no inflammatory reaction, granulation tissue, or hemangioma. The general impression was that of an old hematoma in the process of organization and with poor absorption.

The postoperative course was uneventful, with no symptoms from the facial nerve. For three weeks there was some brownish discharge from the wound, which was purposely kept open with rubber tubing. Eighteen days following the operation inflations of the middle ear were started and continued for one month after the patient's discharge from the hospital June 12, 1954. During this time the drum retained a slight discoloration over the lower part. On the follow-up examinations in January and April, 1955, the eardrum appeared normal. Audiometry revealed an average hearing loss of 20 db., with no change after inflation of the middle ear.

DISCUSSION.

For lack of conclusive evidence of any definite pathology, the etiology of idiopathic hemotympanum still remains in the field of philosophy, mainly dominated by the analogy, which in certain respects exists to serous otitis media, and also by speculations as to how spontaneous hemorrhages may occur in the middle ear cleft, and the blood fail to become absorbed, in contrast to cases with traumatic hemotympanum. Consequently Johnston[6] arrives at the conclusion that somewhere in the middle ear cleft of these patients there is an area of varicosity or even hemangioma, which fails to undergo complete tissue repair, and repeatedly breaks down to permit the escape of fresh blood.

In the above reported case there is the additional feature of a facial nerve paralysis, which appeared at the same time as the ear symptoms. This paralysis might be due to an otitis media, although it did not develop until after the onset of otorrhea; furthermore, the paralysis was complete and persisted longer than is the case when caused by otitis media. One week prior to her symptoms she suffered a skull trauma. The history of a trauma was elicited under direct questioning and it might have been very slight; however, the elapse of one week seems to exclude any direct relationship between the paralysis and this trauma. It seems most natural to explain the paralysis as being a Bell's palsy. How the otorrhea fits in with this is not quite clear; however, on admission the secretions were brownish and viscid and not purulent, as would be expected in an otitis media of long standing Unfortunately, the secretions were not cultured and it is not known whether they were sterile

By now it is generally accepted that most cases of so called Bell's palsy are due to some kind of vascular obstruction around the stylomastoid foramen, causing ischemic changes in the facial nerve, with swelling of the nerve within the bony canal and secondary compression (Audibert et al[1], Cawthorne[2], Sullivan[10], Kettel[7], Hilger[5]) As a further result of the ischemia, in severe cases, exudation, or even a hemorrhage, into the mastoid cells may exist, together with bony necrosis of the walls of the Fallopian canal (Kettel[7], Hall[4], Flodgren[3]) This necrosis is of a purely degenerative type and never inflammatory The present case might fit into this description, except that the secretions were so abundant that some kind of low grade infection must be suspected The partial response to antibiotic therapy, and also the repeated exacerbations, in conjunction with upper respiratory infections, support this view A necrotic area in the mastoid must presumably be very susceptible to infections by way of the middle ear or the blood stream The findings at operation helped to localize the diseased area definitely to the region around the lower portion of the facial canal Histological examination revealed no bony necrosis, however, much time had elapsed since the first acute episode, and such necrosis was hardly to be expected As mentioned, we dared not expose the nerve for fear of recurrence of the paralysis

In the latest report of a case of idiopathic hemotympanum Simonton[9] mentions that his patient suffered from homolateral episodes of hemifacial spasms as well During the radical mastoidectomy he found that the bony wall covering the horizontal portion of the Fallopian canal was absent, and the VIIth nerve in this area was enlarged approximately to four times its normal diameter It might be stated, that at least some cases of hemifacial spasms are brought about by the same changes in the vascular supply around the stylomastoid foramen, as in other instances would produce a Bell's palsy; in fact, some cases do appear following Bell's palsy (Williams, Lambert and Woltman[11], Kettel[8]).

Thus we have two cases of idiopathic hemotympanum with two different concomitant diseases, both pointing towards a common etiology. During the future treatment of such patients it might be well to keep in mind the possibility that a variety of disturbed vascular circulation in the area supplied by the stylomastoid artery may exist.

SUMMARY.

A report is given of a patient with concomitant idiopathic hemotympanum and Bell's palsy, cured by simple mastoidectomy. It is suggested that some cases of idiopathic hemotympanum are due to complete or partial vascular obstruction around the stylomastoid foramen, affecting the nutrition of the facial canal and the surrounding mastoid cells.

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Aabrinken 205.

THE RESPONSIBILITY OF THE DOCTOR TO THE HARD-OF-HEARING PATIENT.

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The hard-of-hearing patient for whom remedial therapy has failed, or for whom it was deemed useless, represents a perplexing problem for the community and a challenging problem for the physician. That these people need effective help is immediately apparent when we consider their handicap. The poignant problem of the deaf has long been recognized by our communities, and has been met by our State educational programs and schools for deaf children.

The acquired deafness in the adult, many of whom are elderly, has not been given more than cursory attention. These deaf do present a social, moral, and almost ethical problem that the doctor of medicine cannot find to be unsurmountable. These people can no longer hold their responsible jobs because of their inability to understand directions sufficiently to make their work reliable, or because the frequent necessity of repetition creates an atmosphere of exasperation. Employers will not and cannot continue to accept the responsibility for the deafened employees' errors or continue to accept the exasperation, with the result that these handicapped persons are discharged from employment and are cast out into an unsympathizing community. These suffering people have been neglected by almost everyone, the doctor in particular.

The deaf try to help themselves by securing hearing aids and only a small fraction are fully satisfied with the aids. Even those who do not become discouraged with their first aid and who have the means and the persistence not to give

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up hope, seldom find anything satisfactory except the cheerful promises of the hearing aid salesperson.

The fact that we do not try to help these people is not altogether our own fault but is the result of various cumulative factors which have inevitably developed in the evolution of the hearing aid and the hearing aid industry. To say that we alone are at fault is flagrantly incorrect; it is time for us to review our positions, correct our errors, and individually help the deaf.

In order to understand our position, we must consider what a hearing aid is and what we can expect from it. A hearing aid is nothing more or less than a miniature sound amplifying system, a small individual public address system. For a hearing aid to do more than one could expect from a public address system would be inconceivable. We theorize that if a deafened person can hear a radio he should be able to hear with its miniature counterpart, a hearing aid. Unfortunately, only a few hearing aids today remotely approach the reproduction of communicable sound by large amplifying systems.

This lack of faithful reproduction in hearing aids represents one of the reasons of propitious importance why we physicians have been passive in recommending hearing aids. A doctor takes legitimate pride in his work, and is constantly trying to achieve something better for his patients. From practical experience in some instances, or from hearsay evidence with his hard-of-hearing patient, the doctor hesitates to recommend even the very best available hearing aid if it does not meet his personal specifications of being more than adaptably useful; furthermore, a doctor is a man of science; he cannot understand why, if sound can be reproduced faithfully in his everyday life in radio, TV., etc., it is not possible to reproduce the same thing in a hearing aid. He will not accept responsibilities for recommending something he believes is not good.

Another reason for our apathy in prescribing hearing aids is the admission of defeat of our sincere therapeutic efforts. When a hearing aid is advised it should not be construed that we have failed medically. We do not hesitate to advise glasses or an artificial limb. In these we know our capabilities and limitations, and do not expect to do the impossible. To admit defeat in deafness does not preclude any concept of further study or trial of methods for restoration of hearing. No. we should not despond, but we should be practical enough to weigh judiciously the evidence at hand, to evaluate the usually remote possibility of restoration of the patient's hearing and to discuss his problem with him.

The evolution of the hearing aid industry very clearly shows another reason for our present status as physicians in the fitting of hearing aids. There is a remarkable parallelism between optic and acoustic appliances. It was not until eye glasses became perfected, and it was not until the basic physics involving refractory errors were known that physicians prescribed glasses. At first the itinerant fitter who had magnifying lenses was the only person who attempted optical correction. It was later when the grinding of lenses became perfected that optometry became a recognized business, based on trial and error correction. Only after the refractive errors could be measured and correlated to the pathology present did doctors accept responsibility for prescribing glasses.

In the hearing aid industry the ear trumpet could hardly interest the scientific man. With the development of electrical acoustic devices better amplification could be produced. At the present time these hearing aids are vastly superior to the initial ones, and at least one make can be compared to the efficiency of glasses. Commercialism has so entered the situation today that most aids have progressed in some respects no further than the lenses sold by itinerant eye-glass peddlers.

It is likewise appalling that the hearing aid industry has not taken it upon itself to educate the doctor more often as to the merits of its product. The pharmaceutical companies spend time and money telling us of their products directly through detail men who inform us of their particular products as well as advancements in general. In a sense we feel responsible for some of these pharmaceutical advancements, because we have more or less demanded better medicines through the detail men, and we have worked together.

The hearing aid industry has not done anything comparable to this. Perhaps if we had closer contact with them we could understand their problems and they could understand what we require. This is not necessarily one sided, for it is our responsibility to understand hearing aids; however, the information available to us from many hearing aid representatives is so meager and so unscientific that it is often worthless. Steps are, however, being made in the direction of the physician-hearing aid industry cooperation. This will make possible mutual understanding, cooperation, and help.

Yet another reason for our not being interested in the fitting of the hearing aid is the lack of our ability to predict much from our audiometric testing. Pure tone audiometry, which is the routine method of office testing, is lamentably inadequate. Pure tone audiometric testing is based upon threshold response at different frequencies. This does not test hearing acuity, and represents only a comparable test of the patient's ability to see the smallest amount of lights of different colors. No ophthalmologist would ever prescribe glasses based on these measurements. Indeed he could not, because he is perfunctorily interested in color perception or threshold vision for correcting optic defects.

Even while there are certain correlations between pure tone audiometry and speech, this paradox in threshold testing persists. Our present routine methods of testing have little if any place in hearing aid fittings. Hearing aids must be and are fitted in reality, without regard to the pure tone threshold audiogram except for the rough acoustic gain required and the flat and high tone emphasis. This is not good. Further investigation and further application of our present knowledge of methods, other than pure tone threshold audiometry, will enable us to predict the applicability of hearing aids for our deafened patients.

From these few factors alone it is easily understood why we have helped the hard-of-hearing adult so little. What can we do about it? The patient comes to us for help, so let us help him in the only direct way we can. We must fit hearing aids. Let us select the best aid for each individual patient. Let us make an impression for the ear piece and see that it fits properly. Let us educate the patient to use his aid in noisy environments by the simple application of simulated noise enviroments in our office with phonograph records; then let us aid in training them to select speech from the non-essential background noise and to understand this speech. Let us give the psychological boost to the patient in order that he does not become discouraged. Finally, let us help the hearing aid industry by finding the best aid and fostering the acceptance of efficient, faithfully reproducing amplifying systems which are forthcoming with development of the electronic industry. Only by doing these things can we fulfill our obligations to our hard-of-hearing patients and to our community.

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Further information and application blanks can be obtained from Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia, 40, Penna.

OSTEOMA OF THE EXTERNAL AUDITORY CANAL, CASE REPORT.*

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MAURICE SELIGMAN, M.D.,
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The rarity of osteoma of the external auditory canal large enough to cause symptoms has been established[1]. O. R. Kline and R. C. Pearce were able to find reports of only 36 cases, and added an additional case. Although some cases have been treated by excision through the external auditory canal, by far the majority have required some type of mastoidectomy. Following is the report of a case treated by excision through the external auditory canal. Preoperative X-ray studies in this case were of considerable help in delineating the extent of the tumor in relation to the middle ear.

The patient was a 44-year-old housewife who had known of a mass in the left ear as long as she could remember. For at least ten years she had had periods of marked hearing loss whenever she got water in the ear. These periods would last from one to two months and would sometimes be accompanied by pain and a thin discharge. In between attacks she noted no loss of hearing.

Examination showed a bony hard tumor occluding the left external auditory canal. The surface was smooth and covered with skin, with its lateral surface deep to the edge of the auricular cartilage. After cleaning off all epithelial debris from the edge of the tumor a very narrow slit was found between the tumor and the canal inferiorly. X-ray in the submento-vertex position showed an indistinct mass in the left external auditory canal with an air space medial to it. It was not possible to get a needle through the slit below the tumor because of pain; however, a tiny flexible polyethylene tube (approximately the size of a No. 23 needle) could be passed beyond the tumor for a depth of 2 cm. Through this tube Diodrast was injected, and X-ray in the Towne position (see Fig. 1) clearly outlined the medial aspect of the tumor and the tympanic membrane. It was then felt that removal through the external auditory canal could be safely accomplished.

At operation on February 1, 1956, the findings bore out the X-ray studies. Using a small dental burr with the contra-angle attachment a small opening was made in the inferior area of the tumor, which was known to be thin. This opening was gradually enlarged until it was

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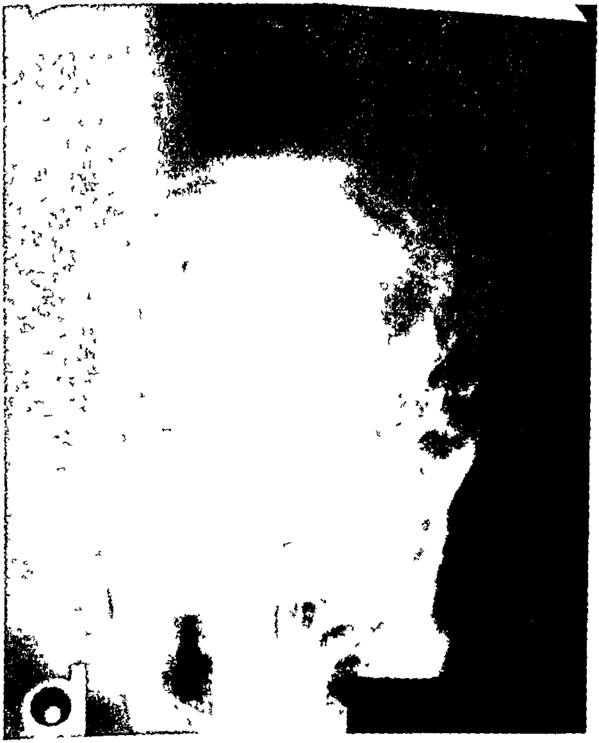

Fig 1 Roentgenogram of mastoids in Towne position showing Diodrast in the air space between the tumor and the tympanic membrane The circular opacity is a lead disc lying against the lateral surface of the tumor

possible to keep the tympanic membrane in view After the canal had been enlarged to about 6 mm the anterior rim of the remaining tumor mass could be identified separate from the antero superior margin of the bony canal wall This rim was engaged with a curette and separated rather easily *en masse* from the canal The surface of the tumor was smooth and covered with connective tissue The canal was of normal contour and the tympanic membrane was intact The tumor measured

8 mm. in its largest diameter and was semilunar in shape (see Fig. 2). Microscopic report was that of a typical osteoma.

Two weeks postoperative the canal was healed and of normal appearance. The audiogram was normal showing no change from the preoperative level.

COMMENT.

Because we were able to get Diodrast between the tumor and the tympanic membrane, it was possible to determine that operation could be safely done through the external auditory canal. Even if a contrast medium could not be introduced it is possible that careful X-ray studies, including laminagrams, could give the same information and sometimes avoid mastoidectomy.

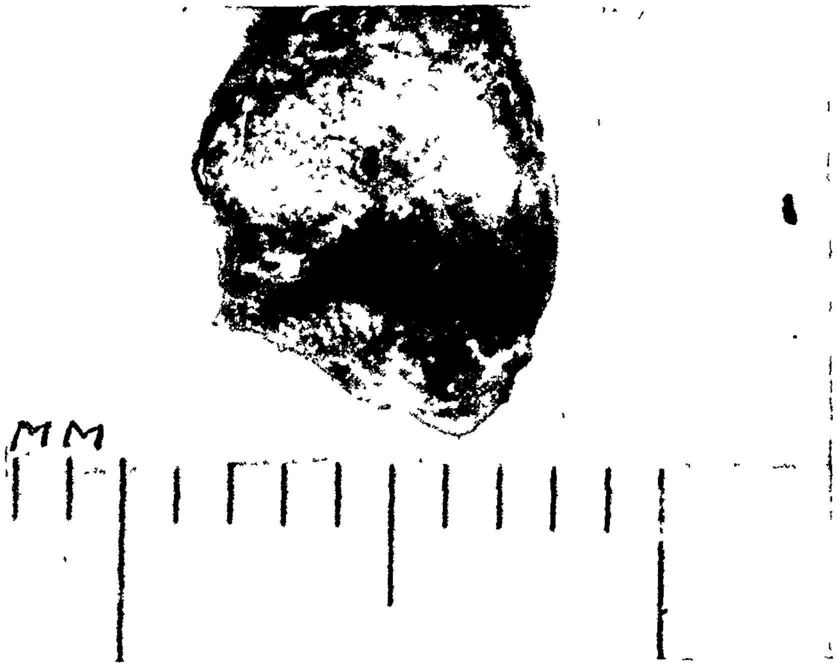

Fig. 2 Tumor after removal.

REFERENCE.

1. Kline, O. R., and Pearce, R. C.: Osteoma of the External Auditory Canal, *Arch. Otol*, 59:588-593, May, 1954.

436 No. Roxbury Drive.

THE RESEARCH STUDY CLUB OF LOS ANGELES

Announces its Twenty-Sixth Annual Mid-Winter Clinical Convention in Ophthalmology and Otolaryngology, January 14 through January 25, 1957.

For "Ear-Nose-Throat," the main Guest Lecturers will include Dr. Louis H. Clerf, Dr. George W. Shambaugh, Jr., and Dr. Maurice H. Cottle.

Mrs. Spencer Tracy, Founder and Director of the John Tracy Clinic of Los Angeles for Deaf and Hard-of-Hearing Children and Their Parents, will speak to the "Ear-Nose-Throat" group the opening day of the Convention on "The Role of Parents in the Education of a Deaf Child."

Dr. H. Russell Fisher, a pathologist of note, will bring us up-to-date in his field.

Instruction Courses in Otolaryngology will be provided by three main speakers, Dr. Clerf, Dr. Shambaugh and Dr. Cottle, as well as by Dr. Aram Glorig, Jr., and Douglas E. Wheeler, Ph. D., both of Los Angeles and authorities in Audiometry.

The main Guest Lecturers for "Eye" program will be Dr. John M. McLean and Dr. Graham Clark, both of New York City; and Dr. Samuel J. Kimura of Berkeley, Calif.

Instruction Courses in Ophthalmology will be conducted by Guest Speaker, Dr. Kimura; Dr. Julian N. Dow, Dr. Robert V. Shearer, Dr. Alfred R. Robbins, and a symposium composed of Dr. Clarence H. Albaugh, Dr. John P. Lordan, and Mr. Russell L. Stimson—all of Los Angeles.

The first week will be devoted to the "Ear-Nose-Throat," from Monday to Friday, January 14 through January 18. The "Eye" week will cover Monday through Friday, January 21 through January 25. Those who confine their work to only one of these specialties may complete either subject in one week, or remain for the full two weeks.

As a period of relaxation, Wednesday afternoons will be

free for sight-seeing, shopping, golf, radio broadcasts, and the enjoyment of points of interest in and about Los Angeles, including the famous Huntington Library with its collection of rare books and art.

Motion pictures of "Ear-Nose-Throat" subjects will be shown during the first week—Monday, Tuesday, Thursday and Friday, 3:00 to 4:00 p.m.

"Eye" subjects will be presented on Wednesday afternoon of the second week for those who prefer to take advantage of this opportunity to view films dealing with subjects in ophthalmology.

The American Laryngological, Rhinological and Otological Society, Western Section, will hold its Annual Meeting in San Francisco between the two weeks of the Convention on Saturday afternoon, January 19, and Sunday morning, January 20. This will permit members to attend the Triologic meeting and return to Los Angeles for the "Eye" week. Programs of the San Francisco meeting will be available at the registration desk at the Elks Club.

On Monday evening, January 21, the annual banquet of the Los Angeles Society of Ophthalmology and Otolaryngology will honor the Guest Lecturers of the "Eye" week. This will take place at 7:00 p. m. in the El Venado Room of the Elks Club.

The Women's Entertainment Committee will arrange for various events of interest to the wives of visiting members during the Convention. This Committee will appreciate information as to whether your wife will attend with you, which will give the Committee an estimate as to the number of wives for whom to plan entertainment. Details of all such events will be printed and available at the registration desk.

Unless you have your own plans for a place to stay, it is advised that you write for reservations at an early date to Mr. R. B. Philbin, Manager of the Elks Club, 607 South Parkview Street, Los Angeles 57. He will endeavor to arrange suitable quarters for you in a conveniently located hotel, and will confirm the reservation by return mail. The Elks Club is the locale for the entire daily program.

All applicants must be members in good standing, of the American Medical Association, *in order to become eligible* for attendance at the Convention. The fee for the entire two weeks, or any part of it, is $100.00 and includes the cost of all luncheons. These dues are an income-tax-deductible item, as they represent an Annual Membership Fee. Make your check payable to "Mid-Winter Clinical Convention," and mail to Norman Jesberg, M.D., Treasurer, 500 South Lucas Avenue, Los Angeles 17.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

Washington, D. C., May 5-10, 1957.

The Officers and the Organizing Committee of the Sixth International Congress of Otolaryngology cordially invite you to attend the Congress which will be held in Washington, D. C., U. S. A., May 5 through 10, 1957.

REGISTRATION AND OPENING.

Any qualified physician may become a Member of the Congress by paying a registration fee of $25.00, U. S. A. Non-medical personnel may register as Associate Members for a fee of $10.00, U. S. A. The registration fee includes the privilege of attending all official meetings of the Congress except the banquet for which an additional charge will be made. Those planning to attend the Congress should complete *Form A* and return it to the General Secretary.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

The meeting dates of the Sixth International Congress of Otolaryngology are again emphasized as May 5th through May 10th, 1957. The scientific program for the Plenary Sessions is now complete and is as follows:

CHRONIC SUPPURATION OF THE TEMPORAL BONE.

OPENERS: Marcus Diamant, Central County Hospital, Halmstad, Sweden—Anatomical Etiological Factors in Chronic Middle Ear Discharge.

Luzius Rüedi, Zurich, Switzerland—Pathogenesis and Treatment of Cholesteatoma in Chronic Suppuration of the Temporal Bone.

Horst Wullstein, Director, Otolaryngological Clinic, University of Würzburg, Germany—Surgical Repair for Improvement of Hearing in Chronic Otitis Media.

DISCUSSERS: A. Tumarkin, Liverpool, England; Juan Manuel Tato, Buenos Aires, Argentina; T. E. Cawthorne, London, England; Fritz Zöllner, Freiburg, Germany.

COLLAGEN DISORDERS OF THE RESPIRATORY TRACT.

OPENERS: Hans Selye, Director, Institute of Experimental Medicine and Surgery, University of Montreal, Faculty of Medicine, Montreal, Canada.

Introduction:

Michele Arslan, Padua, Italy—The Upper Respiratory Tract.

Leslie Gay, Physician-in-Charge, Allergy Clinic, The Johns Hopkins Hospital, Baltimore, U. S. A.—The Lower Respiratory Tract.

DISCUSSERS: Victor E. Negus, London, England; Branimir Gusic, Zagreb, Yugoslavia; Aubrey G. Rawlins, San Francisco, U. S. A.; Henry L. Williams, Rochester, Minn., U. S. A.

PAPILLOMA OF THE LARYNX.

OPENERS: Jo Ono, Tokyo, Japan—Etiology.

Plinio de Mattos Barretto, Faculty of Medicine, University of Sao Paulo, Brazil.

Diagnosis:

F. C. W. Capps, London, England—Therapy.

DISCUSSERS: C. A. Hamberger, Göteborg, Sweden; Pedro Hernandez Gonzalo, Havana, Cuba; Eelco Huizinga, Groningen, Netherlands; Albert von Riccabona, Vienna, Austria.

Anyone planning to attend the Congress and who has not yet registered should do so immediately in order to obtain hotel registration priority.

For more detailed information pertaining to the Sixth International Congress please communicate with the General Secretary, 700 N. Michigan Ave., Chicago 11, Ill., U. S. A.

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DISCUSSERS: Victor E. Negus, London, England; Branimir Gusic, Zagreb, Yugoslavia; Aubrey G. Rawlins, San Francisco, U. S. A.; Henry L. Williams, Rochester, Minn., U. S. A.

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Annual Meeting Palmer House, Chicago, Illinois, October, 1957

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Time and Place Brazil, 1958

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INDEX

SUBJECTS

A

Ablation Therapy for Relief of Meniere's Disease,—Schuknecht H F 859

Acetylcholinesterase Activity in the Cochlea,—Churchill, J A Schuknecht, H F and Doran, R 1

Achondroplastic, Congenital Perichrondritis Associated with,—Kratz, R C 93

A Contribution to the Problem of the Blue Eardrum Idiopathic Hemotympanum, — Terkildsen, K 1582

Adenoid, The, and Its Relation to the Ear,—Alexander, L W 711

A New Method of Bronchoscopy with Cinematography and Photography,—Montreynaud J M de, Edwards, R. J and Gladu, A J 637

A Neurotologic Differentiation Between High and Low Lying Disturbances of the Central Nervous System,—Sellers, L M 935

A Rare Case of Chronic Otorrhea with Intracranial Complications,—Fletcher, R 702

An Evaluation of the Psychogalvanic Skin Resistance Technique in Audiometry,—Bordley, J E 1162

An Otolaryngological Approach to Malignant Exophthalmos,—Boyden, G L 623

An Unusual Case of Eosinophilic Granuloma, — Albano, E H, Aquino, J and Modi, K J 445

APC Viruses in Respiratory Disease—Clinical Aspects,—Parrott, R H 628

Arteries, Large of the Neck, Surgery of the,—Herrmann, L G 1021

A Study of the Role of Certain Factors in the Development of Speech after Laryngectomy Part 1 Type of Operation,—Robe, E Y, Moore, P, Andrews, A H Jr, Holinger, P H 175

A Study of the Role of Certain Factors in the Development of Speech After Laryngectomy Part 2 Site of Pseudoglottis,—Robe, E Y, Moore, P, Andrews, A H Jr, and Holinger, P H 382

A Study of the Role of Certain Factors in the Development of Speech After Laryngectomy Part 3 Coordination of Speech with Respiration,—Robe, E Y, Moore, P, Andrews, A H Jr, and Holinger, P H 481

Antibiotics Part 1 Role of in Acute and Chronic Aural Disorders Part 2 Observations on Children under Two Years of Age Among 50,000 Autopsies at Los Angeles County Hospital Intracranial Complication of Otic Origin,—Hara H J 1049

Atresia of External Auditory Meatus, Congenital,—Goodman M 148

Audiology, Changing Concepts in,—Myklebust, H M 437

A

Auditory Meatus, Congenital Atresia of the External,—Goodman, M. 148

A Variation in Technique for Facial Nerve Decompression,—Lewis, M. L., Jr. 1451

B

Bone, Temporal, Factors Affecting Healing Following Surgery,—Rambo, J. H. T. 810

Bronchoscopy, A New Method of, with Cinematography and Photography,—Montreynaud, J. M. de, Edwards, R. J. and Gladu, A. J. 637

C

Cancer of the Larynx: Neck Dissection, Its Role in,—Reed, G. F. 237

Carcinoma of Larynx, Evolution of Surgical Technique in Treatment of,—Jackson, C. L. and Norris, C. M. 1034

Carcinoma of the Larynx, Surgical Treatment of,—Putney, F. J. and Vicens, E. A. 1041

Changing Concepts in Audiology, —Myklebust, H. M. 437

Children, Rhinology in. Resume and Comments on the Literature for 1955,—Wishart, D. E. S., Whaley, J. B. and Wallace, W. B. 1123

Classroom Demonstration in the Physiology of the Vocal Mechanism,—Boyarsky, L. L. and Kodman, F. 316

Cochlea, Acetylcholinesterase Activity in the,—Churchill, J. A., Schuknecht, H. F. and Doran, R. 1

Congenital Atresia of the External Auditory Meatus,—Goodman, M. 148

Congenital Hemangioma of the Larynx,—Baker, D. C. Jr., and Pennington, C. L. 690

Congenital Perichondritis Associated with Achondroplastic,—Kratz, R. C. 93

Corticosteroids, Review of the—Their Application to Otolaryngology,—Rawlings, A. G. 674

Corti, Organ of, Structures of the Spiral Prominence and External Sulcus and their Relation to the,—Lawrence, M. 796

Dacryocystorhinostomy: Simplified Procedure,—Goodyear, H. M. 152

D

Deaf Children, Educational Guidance for Parents of Young,—Lowell, E. L. and Rushford, G. 1488

Deafness, Development and Adult Anatomy of the Auditory Ossicles in Relation to the Operation for Mobilization of the Stapes in Otosclerotic,—Anson, B. J., and Bast, T. H. 785

Deafness, Middle Ear, Recent Advances in Diagnosis and Treatment of,—Kobrak, H. G. 426

Deafness, of Obscure Origin, Sudden,—Hallberg, O. E. 1237

Deafness, Operation for the Mobilization of the Stapes in Otosclerotic. (Symposium),—Meltzer, P. E., Lindsay, J. R., Goodhill, V., Shambaugh, G. E. Jr., Kos, C. M., Fowler, E. P. Jr., House, H. P., Moderator; Rosen, S., Remarks. With Discussion 729

Defects, Congenital Ossicular, The Recognition and Surgical Treatment of,—Henner, R. and Buckingham, R. A. 526

Development and Adult Anatomy of Auditory Ossicles in Relation to the Operation for Mobilization of the Stapes in Otosclerotic Deafness,—Anson, B. J. and Bast, T. H. 785

agnosis and Treatment of Upper
Respiratory Symptoms Produced
y Increased Viscosity of Mucus
nd Dryness of the Mucous
Membranes,—Anderson, J R.
nd Rubin, W 85

fferential Diagnosis of Hearing
roblems of Adult Patients,—
askins, H L. 638

sease, Meniere's, Ablation Ther
apy for the Relief of,—Schu
necht, H F 859

seases of the Facial Nerve,—
McGovern, F H and Fitz Hugh,
S 187

eases of the Salivary Gland,—
athrop, F D 251

ease, Respiratory—APC Virus
s in,—Parrott, R H 628

orders, Chronic Aural, Intra-
ranial Complications of Otic
rigin Part 1 Role of Antibi
tics in Acute, and Part 2 Ob
ervations on Children under
wo Years of Age Among 50,000
utopsies at Los Angeles County
ospital,—Hara, H J 1049

turbances of the Central Ner
ous System, A Neurotologic
ifferentiation Between High
nd Low Lying,—Sellers L M 935

s, An Experimental Study in,
esigned to Release Tension of
he Vocal Cords in Bilateral Re
urrent Laryngeal Nerve Paral
sis—The Role of the Crico-
hyroid Muscle in Tensions of
e Vocal Cords,—Freedman, L.
574

E

drum, Blue, A Contribution to
e Problem of the Idiopathic
emotympanum, — Terkildsen,
1582

Mesenchyme of the Middle,
he Significance and Fate of
e,—Guggenheim, P, Clements,
and Schlesinger, A 1303

Ear, the, The Adenoid and its Relation to,—Alexander, L W 711

Ear, The, in Head Trauma,—Proctor, B, Gurdjian, E D and Webster, J E 16

Educational Guidance for Parents of Young Deaf Children,—Lowell, E L and Rushford, G 1488

Effectiveness of Conditioned Electrodermal Responses (EDR) in Measuring Pure Tone Thresholds in Cases of Non Organic Hearing Loss,—Goldstein, R. 119

Elongated Styloid Process—Evaluation of Symptoms and Treatment,—Godwin, R W 687

Esophagus, Cervical and Hypopharynx, Reconstruction of the after Radical Excisional Surgery,—Sisson, G A 1268

Evaluation of Surgical Technique in Treatment of Carcinoma of the Larynx,—Jackson, C L and Norris, C M 1034

Exfoliative Cytology of the Larynx—A Preliminary Report,—Whitaker, C W and Seigler, E E 113

Exophthalmos, Malignant, An Otolaryngological Approach to,—Boyden, G L 623

F

Factors Affecting Healing Following Temporal Bone Surgery,—Rambo, J H T 810

Fenestration Operation in the Poor Candidates 44 Cases Selected from 637 Operations,—Farrior, J B 566

G

Gland, Salivary, Diseases of the,—Lathrop, F D 251

Granuloma, Eosinophilic, An Unusual Case of,—Albano, E H, Aquino, J, and Modi, K J 445

H

Hard of Hearing Patient, Responsibility of the Doctor to the,—Forman, F. S. and Graber, A. E. 1587

Headache—A Problem of Modern Otolaryngology,—Sellers, L. M. 131

Headache, Hemicrania or One-Sided Sphenopalatine Ethmoid, —Turnley, W. H. 582

Hearing Center, The Function of a Medical Society-Sponsored,—Newby, H. A. 667

Hearing for Speech, Pure Tone Thresholds for—Diagnostic Significance of Inconsistencies,—Juers, A. L. 402

Hearing Loss and Resultant Audiometric Pattern, Relationship Between Etiology of,—Wilkerson, W. W. Jr., and Doyle, J. I. H. 550

Hearing Losses in Cleft Palate Cases,—Miller, M. H. 1492

Hearing Loss, Identification of by Classroom Teacher,—Kodman, F. Jr. 1346

Hearing, Non-Organic, Effectiveness of Conditioned Electrodermal Responses (EDR) in Measuring Pure Tone Thresholds in Cases of,—Goldstein, R. 119

Hearing Problems of Adult Patients, Differential Diagnosis of, —Haskins, H. L. 658

Hemangioma of Larynx, Congenital,—Baker, D. C. Jr., and Pennington, C. L. 690

Hemicrania—Or One-Sided Sphenopalatine Ethmoid Headache,—Turnley, W. H. 582

Hemorrhage and Controversy, Two Millenia of in Tonsillectomy,—King, J. T. 1068

Hemotympanum, Idiopathic, Review of the Literature and Report on Three Cases,—Sheehy, J. L. and McKibben, B. G. 1291

Hoarseness in Laryngeal Pathology. A Review of the Literature,—Palmer, J. M. 500

Hypopharynx and Cervical Esophagus, Reconstruction of the after Radical Excisional Surgery,—Sisson, G. A. 1268

Hypopharynx, Pulsion Diverticulum of the,—Brintnall, E. S., Grandon, E. L., Lierle, D. M. and Tidrick, R. T. 919

I

Identification of Hearing Loss by Classroom Teacher,—Kodman, F. Jr. 1346

Idiopathic Hemotympanum. Review of the Literature and Report of Three Cases,—Sheehy, J. L. and McKibben, B. G. 1291

Intracranial Complications of Otic Origin: Part 1. Role of Antibiotics in Acute and Chronic Aural Disorders; Part 2. Observations on Children under Two Years of Age among 50,000 Autopsies at Los Angeles County Hospital,—Hara, H. J. 1049

K

Kartagener, Triad of. Relation of Upper to Lower Respiratory Pathology,—Fornatto, E. J., Skolnik, E. M. and Timpkin, R. H. 1202

L

Laryngeal Pathology, Hoarseness in. A Review of the Literature, —Palmer, J. M. 500

Laryngectomy. The Low Collar Incision for Widefield,—Work, W. P. 517

Larynx, Carcinoma of the, Evolution of the Surgical Technique in,—Jackson, C. L. and Norris, C. M. 1034

Larynx, Congenital Hemangioma of the,—Baker, D. C. Jr., and Pennington, C. L. 690

Larynx, Exfoliative Cytology of the—A Preliminary Report,—Whitaker, C W and Siegler, E E 113

Larynx, Human The Mechanism of Opening of the,—Fink, R B, Basek, M and Epanchin, V 410

Larynx, Surgical Treatment of W W 871

Larynx, Papilloma of the,—Webb, Carcinoma of the,—Putney, F J and Vicens, E A 1041

M

Mediastinal Emphysema Complicating Tracheotomy and Thyroidectomy,—Stothers, H E 1411

Medical Otolaryngology,—Hensel, F K 449

Membrane, Tympanic, Repair of Chronic Central Perforations of the, by Repeated Acid Cautery, By Skin Grafting,—Wright, W K 1464

Meningioma, Significance of Otologic Findings in Cerebellopontine,—Brown, H A and Love, J G 1186

Meningioma, Surgical Technique for Removal of Infratemporal,—Conley, J J, Pack, G T and Trinidad, S S 540

Mesenchyme of the Middle Ear, The Significance and Fate of the,—Guggenheim, P, Clements, L and Schlesinger, A 1303

N

Nasal Septum, Surgical Anatomy of the Antero Caudal Portion of the A study of the Area of the Premaxilla,—Klaff, D D 995

Neck Dissection Its Role in Cancer of the Larynx,—Reed, G F 237

Neck, Surgery of the Large Arteries of the,—Herrmann, L G 1021

Nerve Facial, A Variation in Technique for Decompression,—Lewis, M L Jr 1451

Nerve Facial, Diseases of the,—McGovern, F H and Fitz Hugh, G S 187

Nerve Vagus, Tumor of the Ganglion Nodosum of the—Linn, J H and Proctor, B 1577

Nervous System, Central, A Neurotologic Differentiation Between High and Low Lying Disturbances of the,—Sellers, L M 935

O

Odontopathic Aspects in Otolaryngology,—Kowal, R A, Singh, H, and Borkenhagen, R. 1327

Operation, Fenestration, in the Poor Candidates 44 Cases Selected from 637 Operations,—Farrior, J B 566

Ossicles, Auditory, Development and Adult Anatomy of the, in Relation to the Operation for Mobilization of the Stapes in Otosclerotic Deafness,—Anson, B J and Bast, T. H 785

Osteoma of the External Auditory Canal, Case Report,—Seligman, E and Seligman, M 1592

Otitis Media, Serous,—Singleton, J D 293

Otolaryngology, Medical,—Hansel, F K 449

Otolaryngology, Modern, Headache—A Problem of,—Sellers, L. M 131

Otolaryngology, Odontopathic Aspects in,—Kowal, R A, Singh, H, and Borkenhagen, R 1327

Otorrhea, Chronic, A Rare Case of, with Intracranial Complications,—Fletcher, R 702

P

Palate, Cleft, Cases, Hearing Losses in,—Miller, M H 1492

Papilloma of the Larynx,—Webb, W. W. 871

Paralysis, Bilateral Recurrent Laryngeal Nerve, The Role of the Cricothyroid Muscle in Tension of the Vocal Cords. An Experimental Study in Dogs, Designed to Release Tension of the,—Freedman, L. M. 574

Paralysis, Facial, Associated with Fractures of the Temporal Bone,—Maxwell, J. H. and Magielski, J. E. 599

Parotid Gland and Duct, Sialography in the Diagnosis and Treatment of Lesions of the,—Beyer, T. E. and Blair, J. R. 60

Pathology, Respiratory, The Triad of Kartagener. Relation of Upper to Lower,—Fornatto, E. J., Skolnik, E. M. and Timpkin, R. H. 1202

Perichondritis, Congenital, Associated with Achondroplastic, —Kratz, R. C. 93

Pharynx and Pharyngeal Surgery for 1955. Review of the Available Literature on the,—LeJeune, F. E. and Stevenson, E. W. 1383

Premaxilla Area, Surgical Anatomy of the Antero-Caudal Portion of the Nasal Septum, A Study of the,—Klaff, D. D. 995

Present Status of the Operation for Mobilization of Stapes,—Bellucci, R. J. 269

Present Status of Stapedolysis (Stapes Mobilization),—Goodhill, V. 333

Procedure of High Dorsal Rhinoplasty,—Wolfe, M. M. 304

Pseudoglottis, Site of (Part 2). A Study of the Role of Certain Factors in the Development of Speech after Laryngectomy,—Robe, E. Y., Moore, P., Andrews, A. H. Jr., and Holinger, P. H. 382

Pulsion Diverticulum of the Hypopharynx, — Brintnall, E. S., Grandon, E. L., Lierle, D. M., and Tidrick, R. T. 919

Pure Tone Threshold for Hearing for Speech—Diagnostic Significance of Inconsistencies,—Juers, A. L. 402

R

Recent Advances in Diagnosis and Treatment of Middle Ear Deafness,—Kobrak, H. G. 426

Reconstruction of the Hypopharynx and Cervical Esophagus after Radical Excisional Surgery,—Sisson, G. A. 1268

Recruitment Tests at Low Sensation Levels,—Palva, T. 1519

Relationship Between Etiology of Hearing Loss and Resultant Audiometric Pattern,—Wilkerson, W. W. Jr., and Doyle, J. I. H. 550

Repair of Chronic Central Perforations of the Tympanic Membrane by Repeated Acid Cautery; By Skin Grafting,—Wright, W. K. 1464

Responsibility of the Doctor to the Hard of Hearing Patient,—Forman, F. S. and Graber, A. E. 1587

Review of the Available Literature on the Pharynx and Pharyngeal Surgery for 1955,—LeJeune, F. E. and Stevenson, E. W. 1383

Review of the Corticosteroids—Their Application to Otolaryngology,—Rawlings, A. G. 674

Rhinology in Children. Resume and Comments on the Literature for 1955,—Wishart, D. E. S., Whaley, J. B. and Wallace, W. B. 1123

Rhinoplasty, Procedure of High Dorsel,—Wolfe, M. M. 304

S

Serous Otitis Media,—Singleton, J. D 293

Sialography in the Diagnosis and Treatment of Lesions of the Parotid Gland and Duct,—Beyer, T E and Blair, J R 60

Sick People in a Troubled World,—Rusk, H A 1094

Siegle's Pneumatic Ear Speculum Modified for a Manometer A Preliminary Report on Findings, —Moose, R M. 1221

Significance of Otologic Findings in Cerebellopontine Meningioma,—Brown, H A and Love, J. G 1186

Sinusitis, Acute, Treatment of with Penicillin and the Intramuscular Use of Steptokinase, —Miller, J M, Surmonte, J A, Ginsberg, M and Ablondi, F B 143

Sinusitis, Staphylococcus Bacteriophage Lysate Aerosol Therapy of,—Mills, A E 846

Skin Resistance Technique in Audiometry, An Evaluation of the Psychogalvanic,—Bordley, J E 1162

Speculum, Ear, Siegle's Pneumatic, Modified for a Manometer. A Preliminary Report on Findings—Moose, R M 1221

Speech After Laryngectomy, A Study of the Role of Certain Factors in the Development of 1 Type of Operation,—Robe, E Y, Moore, P, Andrews, A H Jr, Holinger, P H 173

Speech After Laryngectomy, A Study of the Role of Certain Factors in the Development of Part 2 Site of Pseudoglottis,—Robe, E Y, Moore, P, Andrews, A H Jr, and Holinger, P 282

Speech After Laryngectomy, A Study of the Role of Certain Factors in the Development of Part 3 Coordination of Speech with Respiration,—Robe, E Y, Moore P, Andrews, A H Jr, and Holinger, P H 481

Spiral Prominence and External Sulcus, Structures of the, and Their Relation to the Organ of Corti—Lawrence, M 796

Stapedolysis, Present Status of (Stapes Mobilization),—Goodhill, V 333

Stapes, Development and Adult Anatomy of the Auditory Ossicles in Relation to the Operation for Mobilization of the, in Otosclerotic Deafness,—Anson, B J and Bast, T H 785

Stapes, Operation for the Mobilization of the, in Otosclerotic Deafness (Symposium),—Meltzer, P E, Lindsay, J R, Goodhill, V, Shambaugh, G E, Kos, C M, Fowler, E P Jr, House, H P Moderator, Rosen S Remarks With Discussion 729

Stapes Mobilization Procedure, Statistical Analysis of the,—Kinney, C E 1339

Stapes, Present Status of the Operation for Mobilization of,—Bellucci, R J 269

Staphylococcus Bacteriophage Lysate Aerosol Therapy of Sinusitis—Mills, A E 846

Statistical Analysis of the Stapes Mobilization Procedure,—Kinney, C E 1339

Strictures of the External Auditory Canal,—Proud, G O'N 72

Structures of the Spiral Prominence and External Sulcus and Their Relation to the Organ of Corti,—Lawrence, M 796

Styloid Process, Elongated—Evaluation of Symptoms and Treatment,—Godwin, R W 687 ymposium: The Operation for the Mobilization of the Stapes, in Otosclerotic Deafness,—Meltzer, P. E., Lindsay, J. R., Goodhill, V., Shambaugh, G. E. Jr., Kos, C. M., Fowler, E. P. Jr., House, H. P. Moderator, Rosen, S., Remarks. With Discussion 729

udden Deafness of Obscure Origin,—Hallberg, O. E. 1237

urgery of the Large Arteries of the Neck,—Herrmann, L. G. 1021

urgery, Temporal Bone, Factors Affecting Healing Following,—Rambo, J. H. T. 810

Surgical Anatomy of the Antero-Caudal Portion of the Nasal Septum. A Study of the Area of the Premaxillary,—Klaff, D. D. 995

Surgical Technique for Removal of Infratemporal Meningioma,—Conley, J. J., Pack, G. T. and Trinidad, S. S. 540

Surgical Treatment of Carcinoma of the Larynx,—Putney, F. J. and Vicens, E. A. 1041

T

Temporal Bone, The Management of Facial Paralysis Associated with Fractures of the,—Maxwell, J. H. and Magielski, J. E. 599

Tests, Recruitment, at Low Sensation Levels,—Palva, T. 1519

The Adenoid and its Relation to the Ear,—Alexander, L. W. 711

The Ear in Head Trauma,—Proctor, B., Gurdjian, E. S. and Webster, J. E. 16

The Function of a Medical Society—Sponsored Hearing Center,—Newby, H. A. 667

The Low Collar Incision for Widefield Laryngectomy,—Work, W. P. 517

The Operation for the Mobilization of the Stapes in Otosclerotic Deafness. (Symposium),—Meltzer, P. E., Lindsay, J. R., Goodhill, V., Shambaugh, G. E. Jr., Kos, C. M., Fowler, E. P. Jr.; House, H. P., Moderator, Rosen, S. Remarks. With Discussion ....729.

The Management of Facial Paralysis Associated with Fractures of the Temporal Bone,—Maxwell, J. H. and Magielski, J. E.....599

The Mechanism of Opening of the Human Larynx,—Fink, R. B., Basek, M. and Epanchin, V. 410

The Problem of Post-Tonsillectomy Poliomyelitis. An Analysis of 888 Cases of Poliomyelitis and 2813 Tonsillectomies from a District in the Southwest United States, 1949-1953,—Engelking, C. F. 1541

The Recognition and Surgical Treatment of Congenital Ossicular Defects,—Henner, R. and Buckingham, R. A. 526

The Role of the Cricothyroid Muscle in Tension of the Vocal Cords. An Experimental Study in Dogs, Designed to Release Tension of the Vocal Cords in Bilateral Recurrent Laryngeal Nerve Paralysis,—Freedman, L. M. 574

The Significance and Fate of the Mesenchyme of the Middle Ear, —Guggenheim, P., Clements, L. and Schlesinger, A. 1303

Theory and Practice of Tympanoplasty,—Wullstein, H. 1076

Tonsillectomy, Post, Poliomyelitis, The Problem of,—Engelking, C. F. 1541

Tonsillectomy: Two Millenia of Hemorrhage and Controversy,—King, J. T. 1068

Tracheotomy and Thyroidectomy, Mediastinal Emphysema Complicating,—Stothers, H. H. 1411

Trauma, Head, The Ear in,—Proctor, B, Gurdjian, E S and Webster, J E 16

Treatment of Acute Sinusitis with Penicillin and the Intramuscular Use of Streptokinase,—Miller J M, Surmonte, J. A, Ginsberg, M, Ablondi, F B 143

Thyroidectomy and Tracheotomy Mediastinal Emphysema Complicating,—Stothers, H H 1411

Triad of Kartagener. Relation of Upper to Lower Respiratory Pathology, — Fornatto, E J, Skolnik, E M and Timpkin, R H 1202

Tumor of the Ganglion Nodosum of the Vagus Nerve,—Linn, J H. and Proctor, B 1577

Tympanoplasty, Theory and Practice of,—Wullstein, H 1076

U

Upper Respiratory Symptoms Produced by Increased Viscosity of Mucus and Dryness of the Mucous *Membranes, The Diagnosis* and Treatment of,—Anderson, J R and Rubin, W 85

V

Viruses, APC in Respiratory Disease—Clinical Aspects,—Parrott, R H 628

Voice Mechanism, Classroom Demonstration in the Physiology of the,—Boyarsky, L L and Kodman, F 316

W

World, Sick People in a Troubled, —Rusk, H A 1094

AUTHORS.

A

ABLONDI, F B, MILLER, J M, GINSBERG, M SURMONTE, J A —Treatment of Acute Sinusitis with Penicillin and the Intramuscular Use of Streptokinase 143

ALBANO, E H, AQUINO, J AND MODI K J —An Unusual Case of *Eosinophilic Granuloma* 445

ALEXANDER, L W —The Adenoid and its Relation to the Ear 711

ANDERSON, J R AND RUBIN, W —Diagnosis and Treatment of Upper Respiratory Symptoms Produced by Increased Viscosity of Mucus and Dryness of the Mucous Membrane 85

ANDREWS, A H JR., ROBE, E Y, MOORE, P, HOLINGER, P H —A Study of the Role of Certain Factors in the Development of Speech After Laryngectomy 1 *Type of Operation* 173

ANDREWS, A H JR, ROBE, E Y, MOORE, P AND HOLINGER, P H —A Study *of the Role of* Certain Factors in the Development of Speech After Laryngectomy Part 2 Site of Pseudoglottis 382

ANDREWS, A H JR, ROBE, E Y, MOORE, P AND HOLINGER, P H —A Study of the Role of Certain *Factors in the Development of* Speech After Laryngectomy Part 3 Coordination of Speech with Respiration 481

ANSON, B J AND BAST, T H —Development and Adult Anatomy of the Auditory Ossicles *in Relation to the Operation for* Mobilization of the Stapes in Otosclerotic Deafness 785

AQUINO, J, ALBANO, E H AND MODI, K J —An Unusual Case of Eosinophilic Granuloma 445

B

Baker, D. C. Jr., and Pennington, C. L.—Congenital Hemangioma of the Larynx 690

Basek, M.; Fink, R. B. and Epanchin, V.—The Mechanism of Opening of the Human Larynx....410

Bast, T. H. and Anson, B. J.—Development and Adult Anatomy of the Auditory Ossicles in Relation to the Operation for Mobilization of the Stapes in Otosclerotic Deafness 785

Bellucci, R. J.—Present Status of the Operation for Mobilization of Stapes 269

Beyer, T. E. and Blair, J. R.—Sialography in the Diagnosis and Treatment of Lesions of the Parotid Gland and Duct 60

Brown, H. A. and Love, J. G.—Significance of Otologic Findings in Cerebellopontine Meningioma 1186

Brintnall, E. S.; Grandon, E. L.; Lierle, D. M. and Tidrick, R. T.—Pulsion Diverticulum of the Hypopharynx 919

Blair, J. R. and Beyer, T. E.—Sialography in the Diagnosis and Treatment of Lesions of the Parotid Gland and Duct 60

Bordley, J. E.—An Evaluation of the Psychogalvanic Skin Resistance Technique in Audiometry 1162

Borkenhagen, R.; Singh, H. and Kowal, R.—Odontopathic Aspects in Otolaryngology 1327

Boyarsky, L. L. and Kodman, L.—Classroom Demonstration in the Physiology of the Vocal Mechanism 316

Boyden, G. L.—An Otolaryngologic Approach to Malignant Exophthalmos 623

Buckingham, R. A. and Henner, R.—The Recognition and Surgical Treatment of Congenital Ossicular Defects 526

C

Churchill, J. A.; Schuknecht, H. F. and Doran, R.—Actylcholinesterase Activity in the Cochlea 1

Clements, L.; Guggenhiem, P. and Schlesinger, A.—The Significance and Fate of the Mesenchyme of the Middle Ear 1303

Conley, J. J.; Pack, G. T. and Trinidad, S. S.—Surgical Technique for Removal of Infratemporal Meningioma 540

D

Doran, R.; Churchill, J. A. and Schuknecht, H. F.—Acetylcholinesterase Activity in the Cochlea 1

Doyle, J. I. H. and Wilkerson, W. W. Jr.—Relationship Between Etiology of Hearing Loss and Resultant Audiometric Pattern....550

E

Edwards, R. J.; Montreynaud, J., M. de, and Gladu, A. J.—A New Method of Bronchoscopy with Cinematography and Photography 637

Engelking, C. F.—The Problem of Post Tonsillectomy Poliomyelitis. An Analysis of 888 Cases of Poliomyelitis and 2813 Tonsillectomies from a District in the Southwest United States, 1949-1953 1541

Epanchin, V.; Fink, R. B. and Basek, M.—The Mechanism of Opening of the Human Larynx....410

F

Farrior, J. B.—Fenestration Operation in the Poor Candidates. 44 Cases Selected From 637 Operations 566

Fink, R. B.; Basek, M. and Epanchin, V.—The Mechanism of Opening of the Human Larynx 410

FITZ-HUGH, G. S. and MCGOVERN, F. H.—Diseases of the Facial Nerve . . . 187

FLETCHER, R.—A Rare Case of Chronic Otorrhea with Intracranial Complications 702

FORMAN, F. S. and GRABER, A. E.—Responsibility of the Doctor to the Hard of Hearing Patient 1587

FORNATTO, E. J., SKOLNIK, E. M. and TIMKIN, R. H.—Triad of Kartagener. Relation of Upper to Lower Respiratory Pathology 1202

FOWLER, E. P. JR.; MELTZER, P. E.; LINDSAY, J. R.; GOODHILL, V.; SHAMBAUGH, G. E. JR.; KOS, C. M.; HOUSE, H. P., Moderator; ROSEN, S., Remarks. Operation for the Mobilization of the Stapes in Otosclerotic Deafness. Symposium with Discussion 729

FRIEDMAN, L. M.—The Role of the Cricothyroid Muscle in Tension of the Vocal Cords. An Experimental Study in Dogs Designed to Release Tension of the Vocal Cords in Bilateral Recurrent Laryngeal Nerve Paralysis 574

G

GOODHILL, V.—Present Status of Stapedolysis (Stapes Mobilization) . . . [illegible]

GOODHILL, V.; MELTZER, P. E.; LINDSAY, J. R.; SHAMBAUGH, G. E. JR.; KOS, C. M.; FOWLER, E. P. JR.; HOUSE, H. P., Moderator; ROSEN, S., Remarks. Operation for the Mobilization of the Stapes in Otosclerotic Deafness. Symposium with Discussion 729

[illegible] Congenital Atresia of the External Auditory Meatus [illegible]

[illegible] Dacryocystorhinostomy [illegible] Procedure [illegible]

GRABER, A. E. and FORMAN, F. S.—Responsibility of the Doctor to the Hard of Hearing Patient 1587

[illegible] of the Hypopharynx [illegible]

[illegible] of the Middle Ear [illegible]

[illegible] Head Trauma [illegible]

B

Baker, D. C. Jr., and Pennington, C. L.—Congenital Hemangioma of the Larynx 690

Basek, M.; Fink, R. B. and Epanchin, V.—The Mechanism of Opening of the Human Larynx....410

Bast, T. H. and Anson, B. J.—Development and Adult Anatomy of the Auditory Ossicles in Relation to the Operation for Mobilization of the Stapes in Otosclerotic Deafness 785

Bellucci, R. J.—Present Status of the Operation for Mobilization of Stapes 269

Beyer, T. E. and Blair, J. R.—Sialography in the Diagnosis and Treatment of Lesions of the Parotid Gland and Duct 60

Brown, H. A. and Love, J. G.—Significance of Otologic Findings in Cerebellopontine Meningioma 1186

Brintnall, E. S.; Grandon, E. L.; Lierle, D. M. and Tidrick, R. T.—Pulsion Diverticulum of the Hypopharynx 919

Blair, J. R. and Beyer, T. E.—Sialography in the Diagnosis and Treatment of Lesions of the Parotid Gland and Duct 60

Bordley, J. E.—An Evaluation of the Psychogalvanic Skin Resistance Technique in Audiometry 1162

Borkenhagen, R.; Singh, H. and Kowal, R.—Odontopathic Aspects in Otolaryngology 1327

Boyarsky, L. L. and Kodman, L.—Classroom Demonstration in the Physiology of the Vocal Mechanism 316

Boyden, G. L.—An Otolaryngologic Approach to Malignant Exophthalmos 623

Buckingham, R. A. and Henner, R.—The Recognition and Surgical Treatment of Congenital Ossicular Defects 526

C

Churchill, J. A.; Schuknecht, H. F. and Doran, R.—Actylcholinesterase Activity in the Cochlea 1

Clements, L.; Guggenhiem, P. and Schlesinger, A.—The Significance and Fate of the Mesenchyme of the Middle Ear 1303

Conley, J. J.; Pack, G. T. and Trinidad, S. S.—Surgical Technique for Removal of Infratemporal Meningioma 540

D

Doran, R.; Churchill, J. A. and Schuknecht, H. F.—Acetylcholinesterase Activity in the Cochlea 1

Doyle, J. I. H. and Wilkerson, W. W. Jr.—Relationship Between Etiology of Hearing Loss and Resultant Audiometric Pattern....550

E

Edwards, R. J.; Montreynaud, J., M. de, and Gladu, A. J.—A New Method of Bronchoscopy with Cinematography and Photography 637

Engelking, C. F.—The Problem of Post Tonsillectomy Poliomyelitis. An Analysis of 888 Cases of Poliomyelitis and 2813 Tonsillectomies from a District in the Southwest United States, 1949-1953 1541

Epanchin, V.; Fink, R. B. and Basek, M.—The Mechanism of Opening of the Human Larynx....410

F

Farrior, J. B.—Fenestration Operation in the Poor Candidates. 44 Cases Selected From 637 Operations 566

Fink, R. B.; Basek, M. and Epanchin, V.—The Mechanism of Opening of the Human Larynx 410

FITZ HUGH, G S and McGOVERN F H—Diseases of the Facial Nerve 187

FLETCHER, R.—A Rare Case of Chronic Otorrhea with Intracranial Complications 702

FORMAN, F S and GRABER A E—Responsibility of the Doctor to the Hard of Hearing Patient 1587

FORNATTO, E J, SKOLNIK E M and TIMKIN, R H—Triad of Kartagener. Relation of Upper to Lower Respiratory Pathology 1202

FOWLER, E P JR, MELTZER P E, LINDSAY, J R, GOODHILL V, SHAMBAUGH, G E JR, KOS C M; HOUSE, H P, Moderator, ROSEN, S, Remarks—Operation for the Mobilization of the Stapes in Otosclerotic Deafness Symposium with Discussion 729

FREEDMAN, L M—The Role of the Cricothyroid Muscle in Tension of the Vocal Cords An Experimental Study in Dogs Designed to Release Tension of the Vocal Cords in Bilateral Recurrent Laryngeal Nerve Paralysis 574

G

GINSBERG, M, MILLER, J M, SURMONTE, J A and ABLONDI, F B Treatment of Acute Sinusitis with Penicillin and the Intramuscular Use of Streptokinase 143

GLADU, A J, MONTREYNAUD J M DE, and EDWARDS, R J—A New Method of Bronchoscopy with Cinematography and Photography 637

GODWIN, R W—Elongated Styloid Process—Evaluation of Symptoms and Treatment 687

GOLDSTEIN, R—Effectiveness of Conditioned Electro Dermal Responses (EDR) in Measuring Pure Tone Thresholds in Cases of Non Organic Hearing Loss 119

GOODHILL, V—Present Status of Stapedolysis (Stapes Mobilization 333

GOODHILL, V, MELTZER P E, LINDSAY J R, SHAMBAUGH, G E JR KOS, C M FOWLER, E P JR, HOUSE H P Moderator ROSEN, S Remarks—Operation for the Mobilization of the Stapes in Otosclerotic Deafness Symposium with Discussion 729

GOODMAN, M—Congenital Atresia of the External Auditory Meatus 148

GOODYEAR H M—Dacryocystorhinostomy—Simplified Procedure 152

GRABER, A E and FORMAN, F S—Responsibility of the Doctor to the Hard of Hearing Patient 1587

GRANDON E L, BRINTNALL E S, LIEBLE, D M and TIDRICK R T—Pulsion Diverticulum of the Hypopharynx 919

GUGGENHEIM, P, CLEMENTS, L and SCHLESINGER, A—Significance and Fate of the Mesenchyme of the Middle Ear 1303

GURDJIAN E S, PROCTOR, B, and WEBSTER, J E—The Ear in Head Trauma 16

H

HALLBERG, O E—Sudden Deafness of Obscure Origin 1237

HANSEL F K—Medical Otolaryngology 449

HARA, H J—Intracranial Complications of Otic Origin Part 1 Role of Antibiotics in Acute and Chronic Aural Disorders Part 2 Observations on Children Under Two Years of Age Among 50,000 Autopsies at Los Angeles County Hospital 1049

HASKINS, H L—Differential Diagnosis of Hearing Problems of Adult Patients

HENNER, H. and BUCKINGHAM, R. A.—The Recognition and Surgical Treatment of Congenital Ossicular Defects 526

HERRMANN, L. G.—Surgery of the Large Arteries of the Neck 1021

HOLINGER, P. H.; ROBE, E. Y.; MOORE, P. and ANDREWS, A. H. JR.—A Study of the Role of Certain Factors in the Development of Speech After Laryngectomy. 1. Type of Operation 173

HOLINGER, P. H.; ROBE, E. Y.; MOORE, P. and ANDREWS, A. H. JR.—A Study of the Role of Certain Factors in the Development of Speech After Laryngectomy. Part 2. Site of Pseudoglottis 382

HOLINGER, P. H.; ROBE, E. Y.; MOORE, P. and ANDREWS, A. H. JR.—A Study of the Role of Certain Factors in the Development of Speech After Laryngectomy. Part 3. Coordination of Speech with Respiration 481

HOUSE, H. P., Moderator; MELTZER, P. E.; LINDSAY, J. R.; GOODHILL, V.; SHAMBAUGH, G. E. JR.; KOS, C. M.; FOWLER, E. P. JR.; ROSEN, S., Remarks—Operation for Mobilization of the Stapes in Otosclerotic Deafness. Symposium with Discussion 729

J

JACKSON, C. L. and NORRIS, C. M.—Evolution of Surgical Technique in Treatment of Carcinoma of the Larynx 1034

JUERS, A. L.—Pure Tone Thresholds for Hearing for Speech—Diagnostic Significance of Inconsistencies 402

K

KING, J. T.—Tonsillectomy: Two Millenia of Hemorrhage and Controversy 1068

KINNEY, C. E.—Statistical Analysis of the Stapes Mobilization Procedure 1339

KLAFF, D. D.—Surgical Anatomy of the Antero-Caudal Portion of the Nasal Septum. A Study of the Area of the Premaxilla 995

KOBRAK, H. G.—Recent Advances in Diagnosis and Treatment of Middle Ear Deafness 426

KODMAN, F. JR.—Identification of Hearing Loss by Classroom Teacher 1346

KODMAN, L. and BOYARSKY, L. L.—Classroom Demonstration in the Physiology of the Vocal Mechanism 316

KOS, C. M.; MELTZER, P. E.; LINDSAY, J. R.; GOODHILL, V.; SHAMBAUGH, G. E. JR.; FOWLER, E. P.; JR.; HOUSE, H. P., Moderator; ROSEN, S., Remarks—Operation for the Mobilization of the Stapes in Otosclerotic Deafness. Symposium with Discussion 729

KOWAL, R. A.; SINGH, H., and BORKENHAGEN, R.—Odontopathic Aspects in Otolaryngology 1327

KRATZ, R. C.—Congenital Perichondritis Associated with Achondroplastic 93

L

LAWRENCE, M.—Structures of the Spiral Prominence and External Sulcus and their Relation to the Organ of Corti 796

LEJEUNE, F. E. and STEVENSON, E. W.—Review of the Available Literature on Pharynx and Pharyngeal Surgery for 1955 1383

LEWIS, M. L. JR.—A Variation in Technique for Facial Nerve Decompression 1451

LIERLE, D. M.; BRINTNALL, E. S.; GRANDON, E. L. and TIDRICK, R. T.—Pulsion Diverticulum of the Hypopharynx 919

[illegible] 129

MILLER, J. M.; SURMONTE, J. A., GINSBERG, M. and ABLONDI, F. B.—Treatment of Acute Sinusitis with Penicillin and the Intramuscular Use of Streptokinase 143

MILLER, M. H.—Hearing Losses in Cleft Palate Cases ... 1192

MILLS, A. E.—Staphylococcus Bacteriophage Lysate Aerosol Therapy of Sinusitis 846

MODI, K. J.; ALBANO, E. H. and AQUINO, J.—An Unusual Case of Eosinophilic Granuloma 445

MONTREYNAUD, JEAN M. DE; EDWARDS, R. J. and GLADU, A. J.—A New Method of Bronchoscopy with Cinematography [illegible] tography

[illegible], B. G. and SHEEHY, J. L.—Idiopathic Hemotympanum Review of the Literature and Report of Three Cases 1291

N

NEWBY, H. A.—The Function of a Medical Society Sponsored Hearing Center 667

NORRIS, C. M. and JACKSON, C. L.—Evolution of Surgical Technique in the Treatment of Carcinoma of the Larynx 1034

P

PACK, G. T., CONLEY, J. J. and TRINIDAD, S. S.—Surgical Technique for Removal of Infratemporal Meningioma 540

P[illegible]

Palva, T.—Recruitment Tests at Low Sensation Levels 1519

Parrott, R. H.—APC Viruses in Respiratory Disease — Clinical Aspects 628

Pennington, C. L. and Baker, D. C. Jr.—Congenital Hemangioma of the Larynx 690

Proctor, B. and Linn, J. H.—Tumor of the Ganglion Nodosum of the Vagus Nerve 1577

Proctor, Bruce; Gurdjian, E. S. and Webster, J. E.—The Ear in Head Trauma 16

Proud, G. O'N.—Stricture of the External Auditory Canal 72

Putney, F. J. and Vicens, E. A.—Surgical Treatment of Carcinoma of the Larynx 1011

R

Rambo, J. H T.—Factors Affecting Healing Following Temporal Bone Surgery 810

Rawlings, A. G.—Review of the Corticosteroids—Their Application to Otolaryngology 674

Reed, G F.—Neck Dissection: Its Role in Cancer of the Larynx 237

Robe, E. Y.; Moore, P.; Andrews, A. H Jr.; Holinger, P. H.—A Study of the Role of Certain Factors in the Development of Speech After Laryngectomy. 1. Type of Operation 173

Robe, E. Y.; Moore, P.; Andrews, A. H. Jr.; Holinger, P. H.—A Study of the Role of Certain Factors in the Development of Speech After Laryngectomy. Part 2. Site of Pseudoglottis 382

Robe, E. Y.; Moore, P.; Andrews, A. H. Jr.; Holinger, P. H.—A Study of the Role of Certain Factors in the Development of Speech After Laryngectomy. Part 3. Coordination of Speech with Respiration 481

Rosen, S., Remarks; Meltzer, P. E.; Lindsay, J. R.; Goodhill, V.; Shambaugh, G. E. Jr.; Kos, C. M.; Fowler, E. P. Jr.; House, H. P., Moderator—Operation for Mobilization of the Stapes in Otosclerotic Deafness. Symposium with Discussion 729

Rubin, W. and Anderson, J. R.—Diagnosis and Treatment of Upper Respiratory Symptoms Produced by Increased Viscosity of Mucus and Dryness of the Mucous Membranes 85

Rushford, G. and Lowell, E. L.—Educational Guidance for Parents of Young Deaf Children 1488

Rusk, H. A.—Sick People in a Troubled World 1091

S

Schlesinger, A.; Clements, L. and Guggenheim, P.—The Significance and Fate of the Mesenchyme of the Middle Ear 1303

Schuknecht, H. F. — Ablation Therapy for Relief of Meniere's Disease 859

Schuknecht, H. F.; Churchill, J. A. and Doran, R.—Acetylcholinesterase Activity in the Cochlea 1

Seligman, E. and Seligman, M.—Osteoma of the External Auditory Canal: Case Report 1592

Seligman, M. and Seligman, E.—Osteoma of the External Auditory Canal: Case Report 1592

Sellers, L. M. — Headache—A Problem of Modern Otolaryngology 131

Sellers, L. M. — A Neurotologic Differentiation Between High and Low Lying Disturbances of the Central Nervous System 935

SHAMBAUGH, G E JR, MELTZER P E, LINDSAY J R, GOODHILL V, KOS, C M, FOWLER E P JR, HOUSE, H P, Moderator, ROSEN, S Remarks—Operation for the Mobilization of the Stapes in Otosclerotic Deafness Symposium and Discussion 729

SHEEHY, J L and McKIBBEN B G—Idiopathic Hemotympanum Review of the Literature and Report of Three Cases 1291

SIEGLER E E and WHITAKER, C W—Exfoliative Cytology of the Larynx—A Preliminary Report 113

SINGLETON J D—Serous Otitis Media 293

SINGH, H, KOWAL R and BORKENHAGEN, R—Odontopathic Aspects of Otolaryngology 1327

SISSON, G A—Reconstruction of the Hypopharynx and Cervical Esophagus After Radical Excisional Surgery 1268

SKOLNIK, E M, FORNATTO E J and TIMPKINS R H—Triad of Kartagener Relation of Upper to Lower Respiratory Pathology 1202

STEVENSON, E W and LEJEUNE, F E—Review of the Available Literature on the Pharynx and Pharyngeal Surgery for 1955 1383

STOTHERS, H H—Mediastinal Emphysema Complicating Tracheotomy and Thyroidectomy 1411

SURMONTE, J A, MILLER, J M, GINSBERG, M, ABLONDI, F B—Treatment of Acute Sinusitis with Penicillin and the Intramuscular Use of Streptokinase 143

T

TERKILDSEN, K—A Contribution to the Problem of the Blue Eardrum Idiopathic Hemotympanum 1582

TIDRICK, R T, BRINTNALL, E S, GRANDON, E L and LIERLE, D M—Pulsion Diverticulum of the Hypopharynx 919

TIMPKIN, R H, FORNATTO, E J, SKOLNIK, E M—Triad of Kartagener Relation of Upper to Lower Respiratory Pathology 1202

TRINIDAD, S S, CONLEY, J J and PACK, G T—Surgical Technique for Removal of Infratemporal Meningioma 540

TURNLEY W H—Hemicrania or One Sided Sphenopalatine Ethmoid Headache 582

V

VIC NS E A and JOHNSON, F J—Surgical Treatment of Carcinoma of the Larynx 1041

W

WALLACE W B, WISHART, D E S and WHALEY, J B—Rhinology in Children Resume and Comments on the Literature for 1955 1123

WEBSTER, J E, PROCTOR, B and GURDJIAN, E S—The Ear in Head Trauma 16

WHALEY, J B, WISHART, D E S and WALLACE W B—Rhinology in Children Resume and Comments on the Literature for 1955 1123

WHITAKER C W and SIEGLER, E E—Exfoliative Cytology of the Larynx—A Preliminary Report 113

WILKERSON, W W JR and DOYLE, J I H—Relationship Between Etiology of Hearing Loss and Resultant Audiometric Pattern 550

WISHART, D E S, WHALEY, J B and WALLACE, W B—Rhinology in Children Resume and Comments on the Literature for 1955 1123

WOLFE M M—Procedure of High Dorsal R 304

WRIGHT, W. K.—Repair of Chronic Central Perforations of the Tympanic Membrane by Repeated Acid Cautery; by Skin Grafting.. 1464

WULLSTEIN, H.—Theory and Practice of Tympanoplasty 1076

IN MEMORIAM

BROWN, JOHN MACKENZIE 98

JONES, ISAAC H. ....1496 and 1503

BOOK REVIEWS

Bronchography in Children, by LUCIO PARENZAN and ALFREDO VAGO 160

L'Education de L'Enfant Sourd, par les parents, avant Ecole, by DRS. MASPETIOL, SOULE, GUILLAMANT, FOURGON and GAUTIE.....464

MISCELLANEOUS

American Laryngological Association, Abtract of Proceedings for 1956 1350

ERRATA 161